The Truth About Cholesterol & Drugs

Dr. Bryan Ardis D.C.

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What is Cholesterol?

Cholesterol 101: An introduction

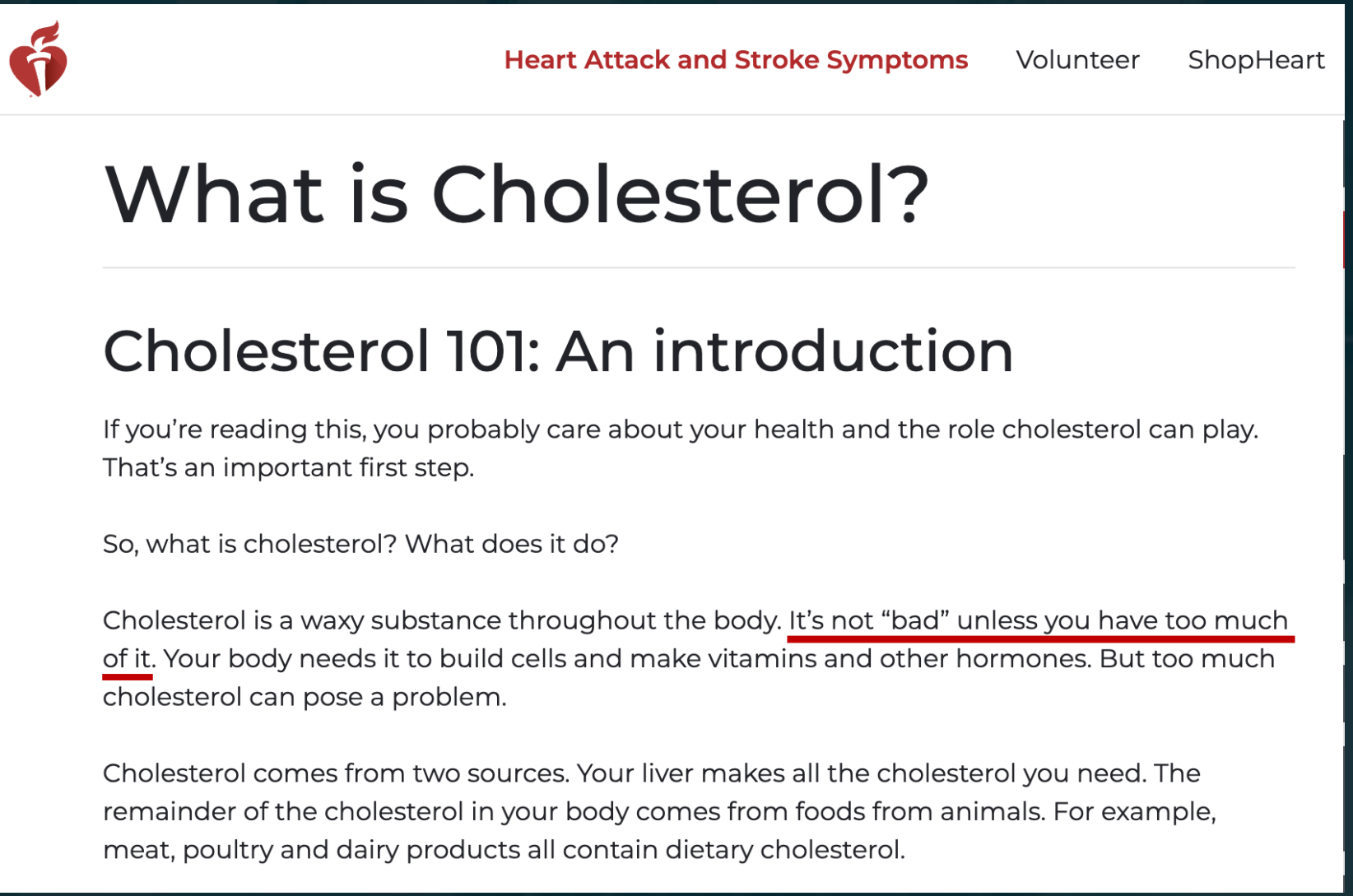
If you're reading this, you probably care about your health and the role cholesterol can play. That's an important first step.

So, what is cholesterol? What does it do?

Cholesterol is a waxy substance throughout the body. It's not "bad" unless you have too much of it. Your body needs it to build cells and make vitamins and other hormones. But too much cholesterol can pose a problem.

Cholesterol comes from two sources. Your liver makes all the cholesterol you need. The remainder of the cholesterol in your body comes from foods from animals. For example, meat, poultry and dairy products all contain dietary cholesterol.

https://www.heart.org/en/health-topics/cholesterol/about-cholesterol



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American Heart Association.

CONSEQUENCES OF HIGH CHOLESTEROL

High cholesterol increases your risk of health issues like heart attack and stroke, the world's top killers. If high cholesterol is left untreated, it can lead to a higher risk or complications with:

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NORMAL ARTERY

CHOLESTEROL BLOCKED ARTERY

Cardiovascular Disease

High cholesterol is a major risk factor for atherosclerotic cardiovascular disease, which is caused by buildup in the arteries and increases the possibility of heart disease and stroke.

Stroke

High amounts of bad cholesterol in your blood can block a critical path to your brain by clogging up your arteries. If the buildup gets too high, it can lead to a stroke.

https://www.heart.org/en/health-topics/cholesterol/about-cholesterol

Diabetes

Diabetes can put a dent in your good cholesterol and increase the amount of bad cholesterol in your blood, increasing your risk of heart attack and stroke.

Artery Disease

PAD (peripheral artery disease) narrows arteries and reduces blood away from your heart to other parts of your body. High blood cholesterol increases your risk of PAD and can also narrow critical pathways through your arteries.

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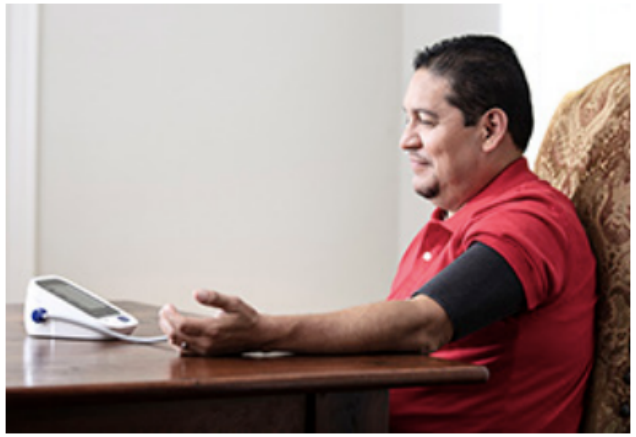

High blood pressure

High blood pressure can damage your blood vessels, where the bad kind of cholesterol (LDL) can build up and narrow your arteries. This can impair your circulatory system and put you at a higher risk for life-threatening conditions.

Lower your cholesterol to lower your risks

Lowering LDL cholesterol will lower your risk of health issues like heart attack and stroke. A first step is checking your cholesterol. Remember lifestyle habits like eating a healthy and balanced diet, moving your body, and eliminating tobacco use can all help you control your cholesterol! Talk to your health care professional about the best treatment plan for you.

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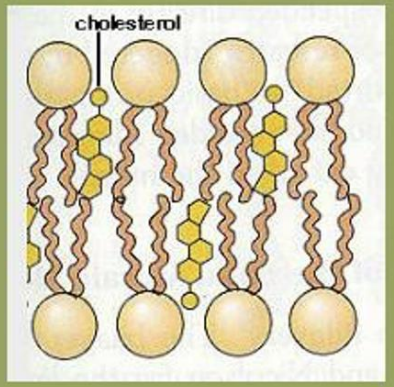
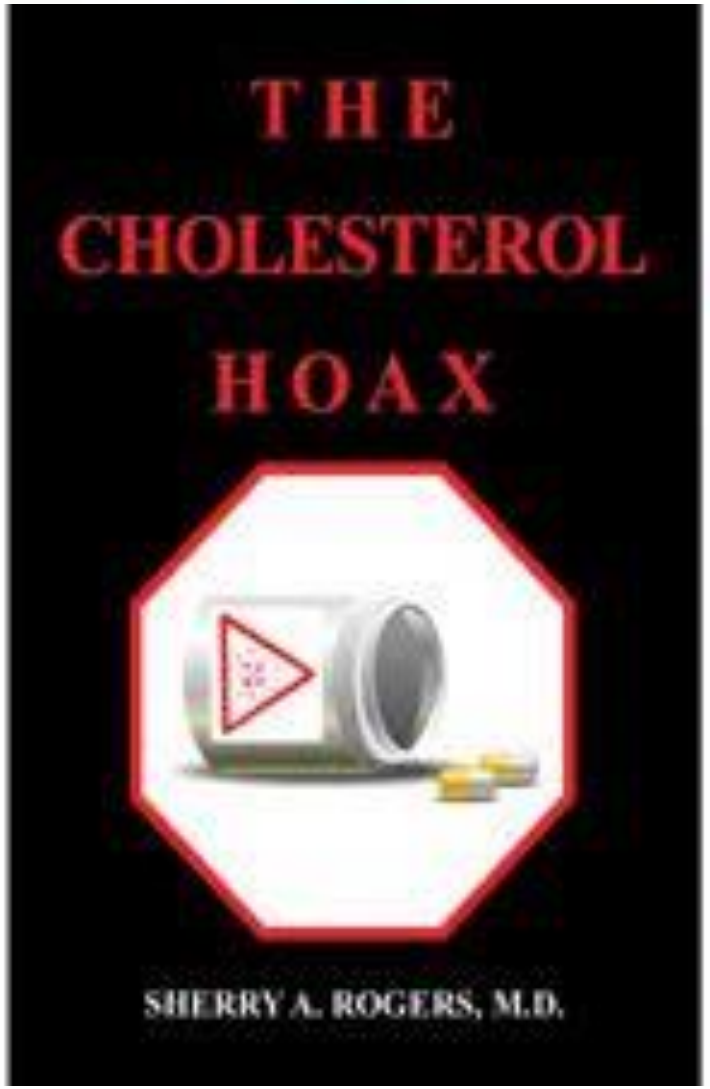
Function of cholesterol



- **is required to build and maintain cell membranes**
- aids in the manufacture of bile (which helps digest fats)
- is important for the metabolism of fat soluble vitamins (vitamins A, D, E and K)
- is the major precursor for the synthesis of vitamin D and of the various steroid hormones (cortisol and aldosterone in the adrenal glands, sex hormones progesterone, estrogen, and testosterone)
- further recent research shows that cholesterol has an important role for the brain synapses as well as in the immune system, including protecting against cancer

https://www.slideserve.com/catori/cholesterol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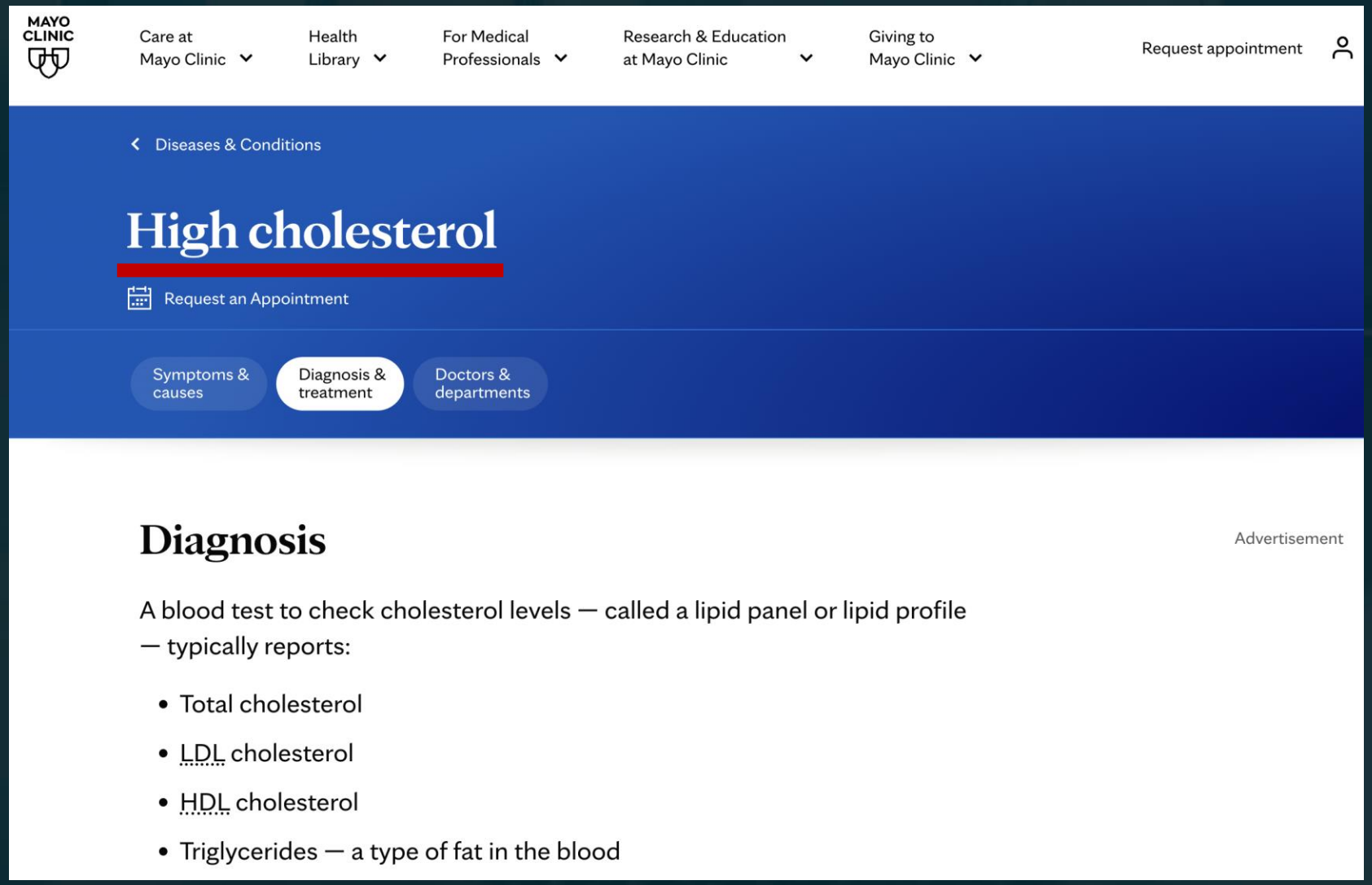

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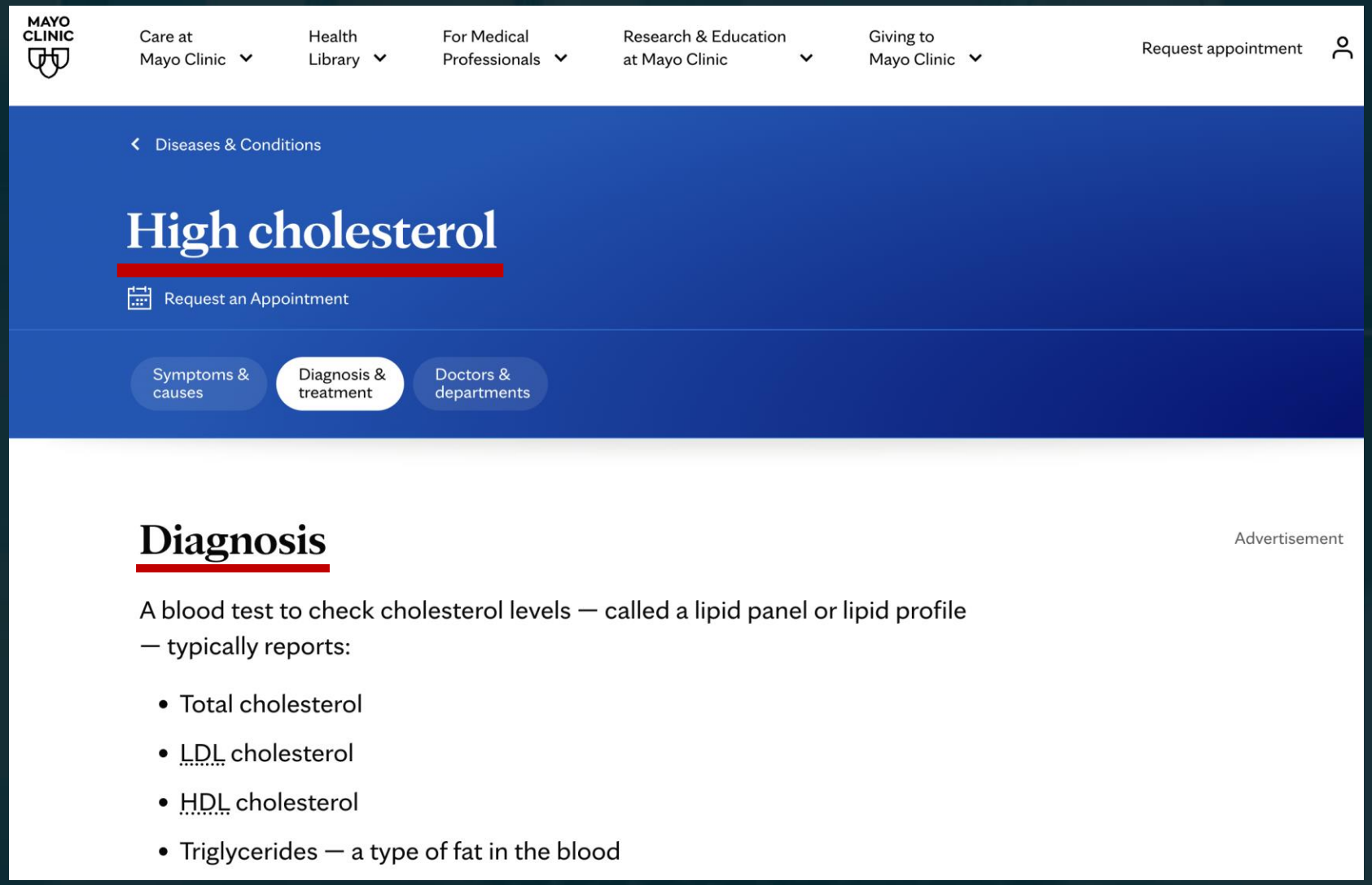

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Interpreting the numbers

In the United States, cholesterol levels are measured in milligrams (mg) of cholesterol per deciliter (dL) of blood. In Canada and many European countries, cholesterol levels are measured in millimoles per liter (mmol/L). To interpret your test results, use these general guidelines.

Total cholesterol (U.S. and some other countries)	Total cholesterol* (Canada and most of Europe)	Results
Below 200 mg/dL	Below 5.2 mmol/L	Desirable
200-239 mg/dL	5.2-6.2 mmol/L	Borderline high
240 mg/dL and above	Above 6.2 mmol/L	High

*Canadian and European guidelines differ slightly from U.S. guidelines. These conversions are based on U.S. guidelines.

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LDL cholesterol (U.S. and some other countries)	LDL cholesterol* (Canada and most of Europe)	Results
Below 70 mg/dL	Below 1.8 mmol/L	Best for people who have coronary artery disease — including a history of heart attacks, angina, stents or coronary bypass.
Below 100 mg/dL	Below 2.6 mmol/L	Optimal for people at risk of coronary artery disease or who have diabetes. Near optimal for people with uncomplicated coronary artery disease.
100-129 mg/dL	2.6-3.3 mmol/L	Near optimal if there is no coronary artery disease. High if there is coronary artery disease.

https://www. c-20350806

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LDL cholesterol (U.S. and some other countries)	LDL cholesterol* (Canada and most of Europe)	Results
130-159 mg/dL	3.4-4.1 mmol/L	Borderline high if there is no coronary artery disease. High if there is coronary artery disease.
160-189 mg/dL	4.1-4.9 mmol/L	High if there is no coronary artery disease. Very high if there is coronary artery disease.
190 mg/dL and above	Above 4.9 mmol/L	Very high, likely representing a genetic condition.

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Interpreting the numbers

HDL cholesterol (U.S. and some other countries)	HDL cholesterol* (Canada and most of Europe)	Results
Below 40 mg/dL (men)	Below 1.0 mmol/L (men)	Poor
Below 50 mg/dL (women)	Below 1.3 mmol/L (women)	
40-59 mg/dL (men)	1.0-1.5 mmol/L (men)	Better
50-59 mg/dL (women)	1.3-1.5 mmol/L (women)	
60 mg/dL and above	Above 1.5 mmol/L	Best

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Interpreting the numbers

Triglycerides (U.S. and some other countries)	Triglycerides* (Canada and most of Europe)	Results
Below 150 mg/dL	Below 1.7 mmol/L	Desirable
150-199 mg/dL	1.7-2.2 mmol/L	Borderline high
200-499 mg/dL	2.3-5.6 mmol/L	High
500 mg/dL and above	Above 5.6 mmol/L	Very high

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Treatment

Lifestyle changes such as exercising and eating a healthy diet are the first line of defense against high cholesterol. But, if you've made these important lifestyle changes and your cholesterol levels remain high, your doctor might recommend medication.

The choice of medication or combination of medications depends on various factors, including your personal risk factors, your age, your health and possible drug side effects. Common choices include:

- **Statins.** Statins block a substance your liver needs to make cholesterol. This causes your liver to remove cholesterol from your blood. Choices include atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Altoprev), pitavastatin (Livalo), pravastatin (Pravachol), rosuvastatin (Crestor) and simvastatin (Zocor).

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- **Cholesterol absorption inhibitors.** Your small intestine absorbs the cholesterol from your diet and releases it into your bloodstream. The drug ezetimibe (Zetia) helps reduce blood cholesterol by limiting the absorption of dietary cholesterol. Ezetimibe can be used with a statin drug.
- **Bempedoic acid.** This newer drug works in much the same way as statins but is less likely to cause muscle pain. Adding bempedoic acid (Nexletol) to a maximum statin dosage can help lower LDL significantly. A combination pill containing both bempedoic acid and ezetimibe (Nexlizet) also is available.

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- **Bile-acid-binding resins.** Your liver uses cholesterol to make bile acids, a substance needed for digestion. The medications cholestyramine (Prevalite), colesevelam (Welchol) and colestipol (Colestid) lower cholesterol indirectly by binding to bile acids. This prompts your liver to use excess cholesterol to make more bile acids, which reduces the level of cholesterol in your blood.
- **PCSK9 inhibitors.** These drugs can help the liver absorb more LDL cholesterol, which lowers the amount of cholesterol circulating in your blood. Alirocumab (Praluent) and evolocumab (Repatha) might be used for people who have a genetic condition that causes very high levels of LDL or in people with a history of coronary disease who have intolerance to statins or other cholesterol medications. They are injected under the skin every few weeks and are expensive.

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Medications for high triglycerides

If you also have high triglycerides, your doctor might prescribe:

- **Fibrates.** The medications fenofibrate (Tricor, Fenoglide, others) and gemfibrozil (Lopid) reduce your liver's production of very-low-density lipoprotein (VLDL) cholesterol and speed the removal of triglycerides from your blood. VLDL cholesterol contains mostly triglycerides. Using fibrates with a statin can increase the risk of statin side effects.
- **Niacin.** Niacin limits your liver's ability to produce LDL and VLDL cholesterol. But niacin doesn't provide additional benefits over statins. Niacin has also been linked to liver damage and strokes, so most doctors now recommend it only for people who can't take statins.
- **Omega-3 fatty acid supplements.** Omega-3 fatty acid supplements can help lower your triglycerides. They are available by prescription or over-the-counter. If you choose to take over-the-counter supplements, get your doctor's OK. Omega-3 fatty acid supplements could affect other medications you're taking.

https://my.clevelandclinic.org/health/treatments/antihyperlipidemic

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Antihyperlipidemic

Antihyperlipidemic medicines can help people bring their cholesterol levels into a normal range. You may need cholesterol-lowering drugs if changing your eating and exercise habits didn't improve your cholesterol numbers. Improving your cholesterol levels can lower your risk of heart attack and stroke.

https://my.clevelandclinic.org/health/treatments/antihyperlipidemic

What are antihyperlipidemic drugs?

Antihyperlipidemic drugs are medicines that help you lower your cholesterol levels. Many people have high cholesterol because of:

- Cholesterol and saturated fat in foods that come from animals (like meat and dairy products) and fried foods (including palm oil products).
- A lack of exercise.
- A genetic issue that causes high cholesterol levels.

Everyone needs some cholesterol in their bodies, but your liver produces the cholesterol you need. The rest is extra, and that excess can lead to atherosclerosis and slow down blood flow in your blood vessels.

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What drugs are antihyperlipidemic?

Types of cholesterol-lowering drugs include:

- Statins (Lipitor®, Crestor®, Zocor® and others).
- PCSK9 inhibitors (Praluent®, Repatha® and Leqvio®).
- Fibric acid derivatives or fibrates (Tricor®, Lopid®, Antara® and others).
- Bile acid sequestrants or bile acid resins (Colestid®, Questran®, Locholest® and others).
- Nicotinic acid or niacin (Niacor® and Niaspan®).
- Selective cholesterol absorption inhibitors (Zetia® and Nexlizet®).
- Omega-3 fatty acids and fatty acid esters (Epanova®, Lovaza® and others).
- Adenosine triphosphate-citrate lyase (ACL) inhibitors (Nexletol® and Nexlizet).

https://my.clevelandclinic.org/health/treatments/antihyperlipidemic

How common are antihyperlipidemic medications?

Antihyperlipidemic medications are very common, especially statins. Tens of millions of people have taken statins since the first one went on the market in 1987.

How do antihyperlipidemics reduce cholesterol?

Different kinds of antihyperlipidemics work in various ways, like:

- Keeping your liver from accessing something it uses to make cholesterol.
- Stopping your body from absorbing cholesterol.
- Slowing down or reducing cholesterol production.
- Helping your liver break down cholesterol.
- Helping your body make more of the “good” cholesterol.

https://my.clevelandclinic.org/health/treatments/antihyperlipidemic

The Truth About
Antihyperlipidemic Drugs!
ardis LABS
www.thedrardisshow.com
THE dr.ardis SHOW

What drugs are antihyperlipidemic?

Types of cholesterol-lowering drugs include:

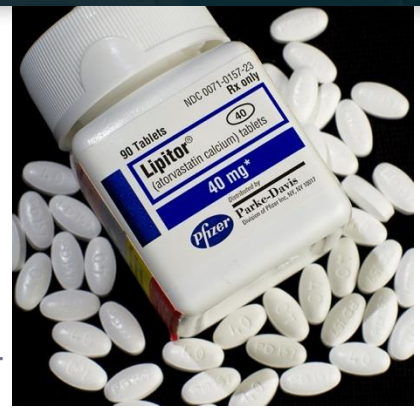
- Statins (Lipitor®, Crestor®, Zocor® and others).
- PCSK9 inhibitors (Praluent®, Repatha® and Leqvio®).
- Fibric acid derivatives or fibrates (Tricor®, Lopid®, Antara® and others).
- Bile acid sequestrants or bile acid resins (Colestid®, Questran®, Locholest® and others).
- Nicotinic acid or niacin (Niacor® and Niaspan®).
- Selective cholesterol absorption inhibitors (Zetia® and Nexlizet®).
- Omega-3 fatty acids and fatty acid esters (Epanova®, Lovaza® and others).
- Adenosine triphosphate-citrate lyase (ACL) inhibitors (Nexletol® and Nexlizet).

https://my.clevelandclinic.org/health/treatments/antihyperlipidemic

Home > Lipitor

Lipitor

Generic name: atorvastatin [*a-TOR-va-sta-tin*]
Dosage form: oral tablet (10 mg; 20 mg; 40 mg; 80 mg)
Drug class: Statins



Medically reviewed by Philip Thornton, DipPharm. Last updated on Oct 12, 2023.

Uses | Warnings | Before taking | Dosage | Side effects | Interactions | FAQ

What is Lipitor?

Lipitor is used together with diet, weight loss, and exercise to reduce the risk of heart attack and stroke and to decrease the chance that heart surgery will be needed in people who have heart disease or who are at risk of developing heart disease.

Lipitor is also used to lower the amount of fatty substances such as low-density lipoprotein (LDL) cholesterol ('bad cholesterol') and triglycerides in the blood and to increase the amount of high-density lipoprotein (HDL) cholesterol ('good cholesterol') in the blood.

Lipitor may also be used to decrease the amount of cholesterol and other fatty substances in the blood in children and teenagers 10 to 17 years of age who have familial heterozygous hypercholesterolemia (an inherited condition in which cholesterol cannot be removed from the body normally).

https://www.drugs.com/lipitor.html

Dermatologic

- **Uncommon** (0.1% to 1%): Rash, pruritus, urticaria, alopecia
- **Rare** (0.01% to 0.1%): Angioedema
- **Postmarketing reports**: Angioneurotic edema, bullous rashes/bullous dermatitis

Bullous rashes/bullous dermatitis included erythema multiforme, Stevens-Johnson syndrome, and toxic epidermal necrolysis.

Endocrine

- **Postmarketing reports**: Gynecomastia

https://www.drugs.com/lipitor.html

Gastrointestinal

- **Very common** (10% or more): Diarrhea (up to 14.1%)
- **Common** (1% to 10%): Dyspepsia, nausea, flatulence, constipation
- **Uncommon** (0.1% to 1%): Abdominal discomfort, abdominal pain, vomiting, upper abdominal pain, lower abdominal pain, pancreatitis, eructation
- **Postmarketing reports**: Pancreatitis

Genitourinary

- **Common** (1% to 10%): Urinary tract infection
- **Uncommon** (0.1% to 1%): Positive for WBCs in urine, erectile dysfunction

Hematologic

- **Postmarketing reports**: Thrombocytopenia

https://www.drugs.com/lipitor.html

Hepatic

- **Common** (1% to 10%): Persistent serum transaminase elevations, abnormal liver function test
- **Uncommon** (0.1% to 1%): Hepatitis
- **Rare** (0.01% to 0.1%): Cholestasis
- **Frequency not reported**: Increased transaminases, abnormal liver function test, jaundice, increased liver enzyme tests, persistent liver enzyme elevations
- **Postmarketing reports**: Fatal/nonfatal hepatic failure

Hypersensitivity

- **Common** (1% to 10%): Allergic reactions
- **Rare** (0.01% to 0.1%): Hypersensitivity
- **Postmarketing reports**: Anaphylaxis

https://www.drugs.com/lipitor.html

Metabolic

- **Common** (1% to 10%): Diabetes, hyperglycemia
- **Uncommon** (0.1% to 1%): Hypoglycemia, anorexia
- **Frequency not reported**: Increased hemoglobin A1c, increased fasting serum glucose levels

Musculoskeletal

- **Very common** (10% or more): Arthralgia (up to 11.7%)
- **Common** (1% to 10%): Pain in extremity, musculoskeletal pain, muscle spasms, myalgia, increased creatine phosphokinase (CPK), joint swelling, back pain
- **Uncommon** (0.1% to 1%): Neck pain, muscle fatigue
- **Rare** (0.01% to 0.1%): Immune-mediated necrotizing myopathy, myositis, myopathy, tendinopathy
- **Postmarketing reports**: Tendon rupture, rhabdomyolysis, lupus-like syndrome, muscle rupture, immune-mediated necrotizing myopathy

https://www.drugs.com/lipitor.html

Nervous system

- **Common** (1% to 10%): Hemorrhagic stroke, headache
- **Uncommon** (0.1% to 1%): Paresthesia, tinnitus
- **Rare** (0.01% to 0.1%): Peripheral neuropathy
- **Postmarketing reports**: Dizziness, peripheral neuropathy, hypoesthesia, amnesia, dysgeusia, myasthenia gravis

Ocular

- **Uncommon** (0.1% to 1%): Blurred vision
- **Rare** (0.01% to 0.1%): Visual disturbance
- **Postmarketing reports**: Ocular myasthenia

https://www.drugs.com/lipitor.html

Other

- **Uncommon** (0.1% to 1%): Deafness/hearing loss, asthenia, malaise, infection, pyrexia
- **Rare** (0.01% to 0.1%): Injury
- **Frequency not reported**: Increased blood alkaline phosphatase
- **Postmarketing reports**: Fatigue, chest pain, peripheral edema, increased weight

Psychiatric

- **Common** (1% to 10%): Insomnia
- **Uncommon** (0.1% to 1%): Nightmare
- **Postmarketing reports**: Depression

Renal

- **Frequency not reported**: Acute kidney injury

https://www.drugs.com/lipitor.html

Respiratory

- **Very common** (10% or more): Nasopharyngitis (up to 12.9%)
- **Common** (1% to 10%): Pharyngolaryngeal pain, epistaxis
- **Uncommon** (0.1% to 1%): Influenza, asthma
- **Rare** (0.01% to 0.1%): Sinusitis, pharyngitis
- **Postmarketing reports**: Interstitial lung disease

https://www.drugs.com/lipitor.html

Home > Crestor

Crestor

Generic name: rosuvastatin [*roe-SOO-va-sta-tin*]
Drug class: Statins

Medically reviewed by Sophia Entringer, PharmD. Last updated on Jan 9, 2025

Uses | Warnings | Before taking | Dosage | Side effects | Interactions | FAQ

What is Crestor?

Crestor belongs to a group of drugs called HMG CoA reductase inhibitors, or "statins." Rosuvastatin is used together with diet to lower blood levels of "bad" cholesterol (low-density lipoprotein, or LDL), to increase levels of "good" cholesterol (high-density lipoprotein, or HDL), and to lower triglycerides (a type of fat in the blood).

Crestor is used in adults and children who are at least 8 years old to lower cholesterol and triglycerides in the blood.

Crestor is used in adults to slow the progression of atherosclerosis (a build-up of plaque in blood vessels that can block blood flow).

https://www.drugs.com/crestor.html

What are some serious side effects of Crestor?

Most medicines may cause some unwanted effects. Although not all of these side effects may occur, if they do occur they may need medical attention.

Serious side effects may include:

- Muscle pain, tenderness and weakness (also called myopathy)
- Acute kidney (renal) failure
- Liver problems
- Depression
- Allergic reactions (hypersensitivity reactions); Angioedema
- Inflammation of the pancreas (pancreatitis)

https://www.drugs.com/crestor.html

What are some serious side effects of Crestor?

Most medicines may cause some unwanted effects. Although not all of these side effects may occur, if they do occur they may need medical attention.

Serious side effects may include:

- Thrombocytopenia (low blood platelet count)
- Weakness, numbness and pain, often in the hands and feet (peripheral neuropathy)
- Scarring of the lung tissue (interstitial lung disease)
- Diabetes mellitus, elevations in HbA1c
- Immune-Mediated Necrotizing Myopathy: cell death in the muscles that cause weakness and fatigue (rare reports)
- Memory loss, forgetfulness, amnesia, memory impairment, and confusion (cognitive impairment). Cognitive impairment has been reported with all statins.

https://www.drugs.com/crestor.html

Muscle pain

Muscle damage with statins can be serious and may rarely cause kidney problems that could lead to death. Contact your doctor right away if:

- you have unexplained muscle pain, tenderness, or weakness, especially with a fever or you feel more tired than usual
- muscle problems even after stopping Crestor (as directed by your doctor).

You may need blood tests to determine the cause of your muscle pain or weakness.

The risk for muscle damage can be higher if you take certain medicines, take higher doses of Crestor, are 65 years of age or older, have uncontrolled thyroid problems (hypothyroidism), or have kidney problems.

https://www.drugs.com/crestor.html

Liver problems

Tell your doctor if you have liver disease or a history of liver disease before you start Crestor treatment. Your doctor should order blood tests to look for any signs of liver damage before you start treatment, and if you develop signs or symptoms of liver injury during treatment.

Call your healthcare provider right away if you:

- feel more tired or weak than usual, lose your appetite
- have pain in your upper belly area
- have dark-colored urine
- notice that your skin or the whites of your eyes turn a yellow color (jaundice).

https://www.drugs.com/crestor.html

Allergic reactions

Do not take this medicine if you are allergic to rosuvastatin calcium, the active ingredient in Crestor.

Serious allergic reactions may include:

- Rash
- Itching
- Hives
- Trouble breathing
- Fast heartbeat
- Redness or flushing
- Swelling under the skin or around the face, eyes, or throat (angioedema)

https://www.drugs.com/crestor.html

Home > Zocor

Zocor

Generic name: simvastatin [*SIM-va-sta-tin*]
Drug class: Statins

Medically reviewed by Sanjai Sinha, MD. Last updated on May 6, 2024.

Uses | Warnings | Before taking | Dosage | Side effects | Interactions | FAQ

What is Zocor?

Zocor belongs to a group of drugs called HMG CoA reductase inhibitors, or "statins."

Zocor is used to lower blood levels of "bad" cholesterol (low-density lipoprotein, or LDL), to increase levels of "good" cholesterol (high-density lipoprotein, or HDL), and to lower triglycerides (a type of fat in the blood).

Zocor is also used to lower the risk of stroke, heart attack, and other heart complications in people with diabetes, coronary heart disease, or other risk factors.

Zocor is used in adults and children who are at least 10 years old.

https://www.drugs.com/zocor.html

Zocor Side Effects

Musculoskeletal

- **Common** (1% to 10%): Myalgia
- **Uncommon** (0.1% to 1%): Myopathy
- **Frequency not reported**: Elevations in creatine kinase, myopathy, dermatomyositis, arthralgia, tendon rupture
- **Postmarketing reports**: Muscle cramps, myalgia, rhabdomyolysis[Ref]

https://www.drugs.com/zocor.html

Zocor Side Effects

Musculoskeletal

- **Common** (1% to 10%): Myalgia
- **Uncommon** (0.1% to 1%): Myopathy
- **Frequency not reported**: Elevations in creatine kinase, myopathy, dermatomyositis, arthralgia, tendon rupture
- **Postmarketing reports**: Muscle cramps, myalgia, rhabdomyolysis[Ref]

Hepatic

- **Common** (1% to 10%): Elevations in liver function tests
- **Frequency not reported**: Hepatitis (including chronic active hepatitis), cholestatic jaundice, fatty changes in the liver, cirrhosis, hepatic failure, fulminant hepatic necrosis
- **Postmarketing reports**: Hepatic failure, hepatitis, jaundice[Ref]

https://www.drugs.com/zocor.html

Gastrointestinal

- **Common** (1% to 10%): Constipation, nausea, flatulence, diarrhea, dyspepsia, abdominal pain, gastritis
- **Frequency not reported**: Anorexia
- **Postmarketing reports**: Pancreatitis, vomiting[Ref]

Hematologic

- **Frequency not reported**: Hemolytic anemia, thrombocytopenia, leucopenia, thrombotic thrombocytopenic purpura (TTP)
- **Postmarketing reports**: Anemia[Ref]

Renal

- **Frequency not reported**: Myoglobinuria, acute renal failure secondary to rhabdomyolysis[Ref]

Cardiovascular

- **Common** (1% to 10%): Angina, edema, atrial fibrillation[Ref]

https://www.drugs.com/zocor.html

Nervous system

- **Common** (1% to 10%): Headache, vertig
- **Frequency not reported**: Cognitive impa
  loss, paraesthesias, peripheral neuropath
- **Postmarketing reports**: Dizziness, pares
  memory loss, forgetfulness, amnesia, me

Dermatologic

- **Common** (1% to 10%): Eczema
- **Frequency not reported**: Pruritic rash, erythema multiform including Stevens-Johnson syndrome, toxic epidermal necrolysis, photosensitivity, purpura
- **Postmarketing reports**: Pruritus, alopecia, a variety of skin changes (e.g., nodules, discoloration, dryness of skin/mucous membranes, changes to hair/nails)[Ref]

https://www.drugs.com/zocor.html

Hypersensitivity

- **Frequency not reported**: Anaphylaxis, angioedema, urticaria, fever, chills, flushing, malaise, dyspnea
- **Postmarketing reports**: Hypersensitivity syndrome which has included some of the follow features: anaphylaxis, angioedema, lupus erythematous-like syndrome, polymyalgia rheumatica, dermatomyositis, vasculitis, purpura, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, eosinophilia, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever, chills, flushing, malaise, dyspnea, toxic epidermal necrolysis, erythema multiforme including Stevens-Johnson syndrome[Ref]

Immunologic

- **Postmarketing reports**: Immune-mediated necrotizing myopathy[Ref]

Ocular

- **Frequency not reported**: Progression of cataracts, ophthalmoplegia[Ref]

https://www.drugs.com/zocor.html

Genitourinary

- **Common** (1% to 10%): Urinary tract infection
- **Frequency not reported**: Impotence, testicular pain, proteinuria
- **Postmarketing reports**: Erectile dysfunction[Ref]

Oncologic

- **Frequency not reported**: Tumor growth, liver, thyroid, lung adenomas, carcinomas[Ref]

Metabolic

- **Common** (1% to 10%): Diabetes mellitus
- **Frequency not reported**: HbA1c and fasting serum glucose levels elevations

Respiratory

- **Common** (1% to 10%): Respiratory tract infection, bronchitis, sinusitis
- **Postmarketing reports**: Interstitial lung disease

https://www.drugs.com/zocor.html

Home > Praluent

Praluent

Pronunciation: *PRAHL-u-ent*
Generic name: alirocumab
Dosage form: Prefilled pen (autoinjector) for subcutaneous use
Drug class: PCSK9 inhibitors

Medically reviewed by Carmen Pope, BPharm. Last updated on Mar 12, 2024.

Uses | Warnings | Before taking | Dosage | Side effects | Interactions | FAQ

What is Praluent?

Praluent (alirocumab) is an injectable medicine that is given under your skin (subcutaneously) which may be used in adults with:

- cardiovascular disease to reduce the risk of heart attack, stroke, and certain types of chest pain conditions (unstable angina) that would normally require hospitalization
- high blood cholesterol (also called primary hyperlipidemia) including an inherited type of high cholesterol called heterozygous familial hypercholesterolemia (HeFH), to reduce low-density lipoprotein cholesterol (LDL-C) or bad cholesterol together with dietary changes. Praluent can be used alone or in addition to other cholesterol-lowering medicines
- another inherited type of high cholesterol called homozygous familial hypercholesterolemia, who require additional lowering of their LDL-C.

Praluent may also be used in children aged 8 years and older with HeFH to reduce LDL-C, alongside dietary changes and other LDL-lowering treatments.

https://www.drugs.com/praluent.html

Hypersensitivity

- **Common** (1% to 10%): Hypersensitivity reaction, nummular eczema, hypersensitivity vasculitis
- **Postmarketing reports:** Angioedema[Ref]

https://www.drugs.com/praluent.html

Hypersensitivity

- **Common** (1% to 10%): Hypersensitivity reaction, nummular eczema, hypersensitivity vasculitis
- **Postmarketing reports**: Angioedema[Ref]

Respiratory

- **Very common** (10% or more): Nasopharyngitis (11.3%)
- **Common** (1% to 10%): Influenza, bronchitis, sinusitis, cough[Ref]

Genitourinary

- **Common** (1% to 10%): Urinary tract infection[Ref]

Hepatic

- **Common** (1% to 10%): Elevated liver enzymes[Ref]

https://www.drugs.com/praluent.html

Home > TriCor

TriCor

Generic name: fenofibrate [*FEN-oh-FYE-brate*]
Drug class: Fibric acid derivatives

Medically reviewed by Philip Thornton, DipPharm. Last updated on Aug 20, 2023.

Uses | Warnings | Before taking | Dosage | Side effects | Interactions

What is TriCor?

TriCor belongs to a group of drugs known as "fibrates." It helps reduce cholesterol and triglycerides (fatty acids) in the blood. High levels of these types of fat in the blood are associated with an increased risk of atherosclerosis (clogged arteries).

TriCor is used along with a proper diet to help to treat high cholesterol and high triglyceride levels.

https://www.drugs.com/tricor.html

Serious side effects of TriCor

- **Common** (1% to 10%): Development of antidrug antibody (ADA), neutralizing antibodies (NAb)
- **Postmarketing reports**: Flu-like illness[Ref]

Musculoskeletal

- **Common** (1% to 10%): Myalgia, muscle spasm, contusion, musculoskeletal pain[Ref]

Nervous system

- **Uncommon** (0.1% to 1%): Neurocognitive events, confusion or memory impairment[Ref]

Metabolic

- **Frequency not reported**: Low LDL-C values

https://www.drugs.com/praluent.html

Serious side effects of TriCor

Hepatic

- **Common** (1% to 10%): Liver function tests abnormal, AST increased, ALT increased
- **Uncommon** (0.1% to 1%): Cholelithiasis
- **Rare** (less than 0.1%): Hepatitis
- **Postmarketing reports**: Cirrhosis, liver fatty deposit, cholecystitis, jaundice, cholangitis, biliary colic, elevated total bilirubin, serious drug-induced liver injury including liver transplantation and death[Ref]

Respiratory

- **Very common** (10% or more): Rhinitis (up to 23.2%), bronchitis (16.4%)
- **Common** (1% to 10%): Respiratory disorder, pharyngitis, sinusitis
- **Uncommon** (0.1% to 1%): Pulmonary embolism
- **Very rare** (less than 0.01%): Interstitial pneumopathy
- **Frequency not reported**: Cough increased, dyspnea, asthma, pneumonia, laryngitis
- **Postmarketing reports**: Interstitial lung disease[Ref]

https://www.drugs.com/tricor.html

Musculoskeletal

- **Very common** (10% or more): Arthralgia (up to 25.1%), back pain (up to 15%)
- **Common** (1% to 10%): Back pain, myalgia
- **Uncommon** (0.1% to 1%): Diffuse myalgia, myositis, muscle cramp, muscle weakness
- **Very rare** (less than 0.01%): Rhabdomyolysis
- **Frequency not reported**: Arthritis, tenosynovitis, joint disorder, arthrosis, leg cramp, bursitis, myasthenia
- **Postmarketing reports**: Muscle spasm[Ref]

Gastrointestinal

- **Very common** (10% or more): Abdominal pain (up to 15.9%)
- **Common** (1% to 10%): Nausea, constipation, diarrhea, vomiting, flatulence
- **Uncommon** (0.1% to 1%): Pancreatitis
- **Frequency not reported**: Dyspepsia, gastroenteritis, rectal disorder, esophagitis, gastritis, colitis, tooth disorder, gastrointestinal disorder, duodenal ulcer, peptic ulcer, rectal hemorrhage, eructation, gamma glutamyl transpeptidase, dry mouth[Ref]

https://www.drugs.com/tricor.html

Nervous system

- **Common** (1% to 10%): Headache, dizziness
- **Rare** (less than 0.1%): Peripheral neuropathy
- **Frequency not reported**: Migraine, paresthesia, hype

Dermatologic

- **Common** (1% to 10%): Rash, pruritus, urticaria
- **Uncommon** (0.1% to 1%): Photosensitivity reaction
- **Rare** (0.01% to 0.1%): Alopecia
- **Very rare** (less than 0.01%): Cutaneous photosensitivity with erythema, vesiculation, nodulation
- **Frequency not reported**: Erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis, eczema, acne, sweating, fungal dermatitis, skin disorder, contact dermatitis, maculopapular rash, nail disorder, skin ulcer
- **Postmarketing reports**: Severe cutaneous adverse drug reactions (SCAR), including Stevens-Johnson Syndrome, Toxic Epidermal Necrolysis, and Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), ecchymosis[Ref]

https://www.drugs.com/tricor.html

Cardiovascular

- **Uncommon** (0.1% to 1%): Deep vein thrombosis
- **Frequency not reported**: Angina pectoris, hypertension, vasodilatation, coronary artery disorder, electrocardiogram abnormal, extrasystole, myocardial infarct, peripheral vascular disorder, varicose vein, cardiovascular disorder, hypotension, palpitation, arrhythmia, phlebitis, tachycardia, atrial fibrillation[Ref]

Ocular

- **Common** (1% to 10%): Conjunctivitis
- **Frequency not reported**: Eye disorder, amblyo fied, refraction disorder[Ref]

Hematologic

- **Rare** (less than 0.1%): Hemoglobin decreased, leukopenia
- **Frequency not reported**: Eosinophilia, lymphadenopathy, thrombocytopenia, anemia
- **Postmarketing reports**: Hematocrit decreased[Ref]

https://www.drugs.com/tricor.html

Other

- **Very common** (10% or more): Accidental injury (up to 18.4%), flu syndrome (up to 17.9%), chest pain (up to 10.1%)
- **Common** (1% to 10%): Creatine phosphokinase increased, malaise, vertigo, fatigue
- **Uncommon** (0.1% to 1%): Serum creatinine increased, urea increased
- **Rare** (less than 0.1%): Sexual asthenia
- **Frequency not reported**: Pain, infection, cyst, hernia, fever, peripheral edema, ear pain, otitis media, herpes zoster, herpes simplex, weight gain, weight loss, edema
- **Postmarketing reports**: Asthenia[Ref]

Genitourinary

- **Uncommon** (0.1% to 1%): Sexual dysfunction
- **Frequency not reported**: Urinary frequency, prostatic disorder, dysuria, gynecomastia, vaginal moniliasis, cystitis[Ref]

https://www.drugs.com/tricor.html

Metabolic

- **Frequency not reported**: Appetite increased, anorexia, diabetes mellitus, hypoglycemia, gout, hyperuricemia
- **Postmarketing reports**: High-density lipoprotein cholesterol levels severely depressed[Ref]

Renal

- **Frequency not reported**: Kidney function abnormal, urolithiasis
- **Postmarketing reports**: Renal failure, acute renal failure[Ref]

Psychiatric

- **Common** (1% to 10%): Insomnia
- **Frequency not reported**: Depression, libido decreased, anxiety, nervousness[Ref]

Endocrine

- **Frequency not reported**: Diabetes mellitus[Ref]

https://www.drugs.com/tricor.html

How To Naturally Correct Cholesterol!

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Lipids in Health and Disease

Lipids Health Dis. 2020 May 28;19:112. doi: 10.1186/s12944-020-01298-4

The effects of combined magnesium and zinc supplementation on metabolic status in patients with type 2 diabetes mellitus and coronary heart disease

Zahra Hamedifard [1], Alireza Farrokhian [2], Željko Reiner [3], Fereshteh Bahmani [1], Zatollah Asemi [1], Maryam Ghotbi [1], Mohsen Taghizadeh [1,✉]

Author information Article notes Copyright and License information

PMCID: PMC7257447 PMID: 32466773

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https://pmc.ncbi.nlm.nih.gov/articles/PMC7257447/

Research design

Patients were randomly assigned into 2 treatment groups (after strafication based on baseline BMI and age): one taking 250 mg per day magnesium (magnesium oxide) and 150 mg/day zinc (zinc sulfate) containing 30 mg elementary zinc or placebo (starch) (Barij Essence; Kashan: Iran) ($n = 30$ in each group) for 12 weeks. Due to the lack of evidence

https://pmc.ncbi.nlm.nih.gov/articles/PMC7257447/

Magnesium and zinc co-supplementations significantly reduced FPG (β − 9.44 mg/dL, 95% CI, − 18.30, − 0.57; $P = 0.03$) and insulin levels (β − 1.37 µIU/mL, 95% CI, − 2.57, − 0.18; $P = 0.02$), and significantly increased the levels of HDL-cholesterol (β 2.09 mg/dL, 95% CI, 0.05, 4.13; $P = 0.04$) when compared with the placebo group (Table 2). Magnesium plus zinc taking was associated with a significant decrease in CRP (β − 0.85 mg/L, 95% CI, − 1.26, − 0.45; $P < 0.001$), and a significant increase in total nitrite (β 5.13 µmol/L, 95% CI, 1.85, 8.41; $P = 0.003$) and TAC (β 43.44 mmol/L, 95% CI, 3.39, 83.50; $P = 0.03$) when compared with the placebo group. Magnesium and zinc co-supplementation also significantly decreased BDI (β − 1.66; 95% CI, − 3.32, − 0.009; $P = 0.04$) and BAI scores (β − 1.30, 95% CI, − 2.43, − 0.16; $P = 0.02$) when compared with the placebo group. Magnesium and zinc co-supplementation did not have any significant effects on the other metabolic parameters when compared with the placebo group.

https://pmc.ncbi.nlm.nih.gov/articles/PMC7257447/

Impact of magnesium plus zinc supplementation on glycemic control and lipoproteins

This study showed that magnesium plus zinc supplements in subjects with CHD and T2DM during 12 weeks caused a significant decrease in FPG and insulin levels, and significant increase in HDL-cholesterol, but it had no effect on the HOMA-IR, QUICKI, total cholesterol, triglycerides, VLDL-cholesterol and LDL-cholesterol levels. According to previous studies, chronic hyperglycemia and dyslipidemia due to increasing insulin resistance and inflammatory cytokines increase the risk of diabetic and atherogenic complications [34, 35]. Earlier, it was reported that nutritional supplements have the beneficial effects on metabolic profiles [8, 36]. A meta-analysis indicated that magnesium intake is associated with a significant improvement in FPG, triglycerides, HDL-cholesterol and LDL-cholesterol levels

https://pmc.ncbi.nlm.nih.gov/articles/PMC7257447/

[37]. In a study on pregnant women with GDM who were taking as a supplement magnesium oxide (250 mg/day) during 6 weeks, a significant improvement in the FPG, insulin concentration, HOMA-IR, QUICKI, and triglycerides levels occurred, but it caused no change in other serum lipoproteins [7]. However, magnesium lactate in a dose of 360 mg per day during 12 weeks did not have significant effects on HbA1c, FPG, insulin levels, HOMA-IR and lipid profiles in normomagnesemic patients with T2DM [38]. One meta-analysis showed that supplementing zinc to patients with T2DM has been associated with a considerable decrease of FPG, HbA1c, total cholesterol, and a significant increase of HDL-cholesterol, but there has been no significant association with triglycerides [39]. Another study performed by Islam et al. showed that zinc supplementation (30 mg per day of zinc sulphate) during 6 months improved FPG, insulin resistance, insulin sensitivity and decreased triglycerides levels without causing any significant changes in LDL-cholesterol and HDL-cholesterol [10].

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A combination of magnesium-zinc-calcium-vitamin D during a 12 week period has been associated with a significant decrease of HOMA-IR, insulin, triglycerides, total cholesterol, and VLDL-cholesterol and a significant increase in the QUICKI scores, but it has not been accompanied by changes in LDL-cholesterol, FPG, and HDL-cholesterol in the PCOS patients [14]. Hyper-insulinemia and insulin resistance have been related to hyperglycemia which is the main symptom of T2DM. Hyperglycemia affects the glycation of lipoproteins but has a lot of other unfavorable effects which cause accelerated atherosclerosis [40, 41]. Magnesium is one of the crucial cofactors in the enzymatic processes that require adenosine triphosphate and kinase, and therefore it plays an important role in glucose metabolic pathways [15, 42]. Magnesium also contributes to lipoprotein metabolism by modulating HMG-CoA reductase enzyme [16]. Zinc is involved in a range of functions including insulin receptor signal

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enzyme [16]. Zinc is involved in a range of functions including insulin receptor signal transduction, secretion and tissues/organelle distribution, and inhibition of protein tyrosine phosphatases [17, 43, 44]. Zinc may increase glucose transport into the cells by increasing phosphorylation of the β subunit of the insulin receptor and enhancing the activation of phosphatidylinositol 3 kinase and protein kinase B or Akt [45]. Furthermore, few zinc transporters (such as ZnT8) are important for the structure, secretion and compartmentalization of insulin in beta-cells of pancreas [46]. Zinc also stimulates IRAP molecule, which in turn enables the translocation of GLUT4 to the cell surface and enables the transport of glucose into the cell [47]. Zinc plays an important role in the stabilization of insulin hexamers and the pancreatic storage of insulin as well [43].

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Effects of magnesium plus zinc supplementation on oxidative stress and inflammation

This study showed a significant decrease of CRP and a significant increase of TAC and total nitrite as a result of combined magnesium and zinc supplementation to the patients with CHD and T2DM during a 12 weeks period. Oxidative stress and inflammation because of developing micro- and macrovascular complications are important risk factors for diabetes and diabetes-associated atherosclerosis [48]. Asemi et al. study indicated that magnesium supplement.

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(250 mg/day of magnesium oxide) in pregnant women with GDM during 6 weeks significantly decreased hs-CRP, but there were no significant changes in TAC and GSH concentrations [7]. Nevertheless, magnesium supplement (magnesium oxide) in a dose of 250 mg per day during 8 weeks to overweight women did not cause any significant changes in inflammatory markers [49]. In another study, taking zinc supplement (30 mg per day of zinc sulphate) during 6 months did not cause any significant change in CRP in women with pre-diabetes [10]. However, magnesium-zinc-calcium-vitamin D supplementation for 12 weeks in women with PCOS was associated with a decrease in CRP levels [14]. Combined

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magnesium-zinc-calcium-vitamin D supplementation in another similar study caused a significant decrease in hs-CRP and MDA, and an increase in TAC without any significant changes in nitric oxide (NO) and GSH levels [50]. A recent study reported that combined magnesium and zinc supplementation (250 mg/day of magnesium oxide plus 220 mg/day zinc sulfate) to women with PCOS during 12 weeks improved hs-CRP and TAC although no significant effects were been seen on NO, MDA and GSH levels [13]. However, these discrepancy between studies may be due to differences in sampling method, study duration, age ranges, dosage of magnesium and zinc used, the characteristics of participants, differences between intervention and control groups, cross-over design or parallel design, and dietary intake of participants.

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Conclusions

The combined magnesium and zinc supplementation during 12 weeks had beneficial effects on FPG, insulin, total nitrite, HDL-cholesterol, CRP, TAC levels, BDI and BAI score. However, it had no significant impact on other metabolic variables in patients with T2DM and CVD. This suggests that magnesium and zinc co-supplementation may be beneficial for patients with T2DM and CHD. Further research is needed on more patients and for longer periods to determine the safety of magnesium and zinc co-supplementation.

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Endocrinol Diabetes Metab. 2024 Oct 15;7(6):e511. doi: 10.1002/edm2.511

Comparative Efficacy of Magnesium and Potassium Towards Cholesterol and Quality of Life in Patients With Type 2 Diabetes Mellitus: A Randomised Single-Blinded Controlled Clinical Trial

Sidra Khalid [1,✉], Riffat Mehboob [2,3,✉], Syeda Shazia Bokhari [4], Muhammad Ali [2], Ambreen Shabbir [2], Khurram Mehboob [2], Hafsa Adnan [2], Mohammed Matoog Karami [5], Hani Shalabi [6], Bander Alshehri [6]

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2.7. Study Groups and Supplementation

To test the effects of potassium and magnesium supplements on cholesterol in patients with T2DM, four groups were formed that were exposed to medical treatment for 60 days. Group I (T1) was the control/placebo group that consisted of patients who received starch (Calip D 250 mg × 2) supplements; Group II (T2) received magnesium supplements (Ostin 250 mg × 2); Group III (T3) received potassium supplements (Paravit 250 mg × 2); Group IV (T4) received both Mg and K medication in form of Bionta (250 mg × 2). Different tests were

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T1: Control

T2: Magnesium

T3: Potassium

T4: MG & K

TABLE 2.

Medication group-based differences in cholesterol levels.

Groups	*N* (%)	Cholesterol I	Cholesterol II	*p* value
T1	68 (23.45)	217.6 ± 68	183.2 ± 68.55	0.003
T2	75 (25.8%)	211 ± 65.41	171 ± 52.69	0.00006
T3	74 (25.5%)	224.9 ± 61.92	163.4 ± 48.38	<0.000001
T4	73 (25.1%)	222.5 ± 49.06	196.2 ± 54.06	0.002495
p value		**0.5277**	**0.0028**	

Note: Bold value is describing the difference among treatment groups.

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The treatment had a major impact on cholesterol levels in all the four groups. Specifically, there was a decrease in mean cholesterol levels in all the groups after the treatment, with the largest reduction seen in T4 group. Similar results were found by Eriksson J et al., who studied the effect of ascorbic acid and magnesium supplements in 56 patients with diabetes [22]. They observed beneficial effects of above-mentioned supplements on triglycerides and cholesterol levels ($p < 0.05$) in patients with diabetes. The research concluded a significant increase in the social QoL of patients with diabetes. The p value was 0.000, which is more significant. Our results confirm past research showing the efficacy of magnesium in the management of stress, anxiety and depression [23, 24, 25, 26, 27, 28, 29].

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In conclusion, this investigation looked at the impact of magnesium and potassium supplements on cholesterol levels and QoL in patients with type 2 diabetes. The treatment had a significant effect on cholesterol levels, with the largest reduction seen in the group that received potassium supplement. The study showed a significant increase in the social QoL after medication followed by the effect on the psychological domain of QoL that needs explanation beyond the pre-set symptoms. These findings suggest that magnesium and potassium supplements may be beneficial in improving the lipid profile and overall QoL of patients with T2DM. Further studies are required with larger sample sizes to enhance the validity of findings. Since the duration of intervention was 60 days in this study, a longer follow-up is required to assess long-term effectiveness of potassium and magnesium.

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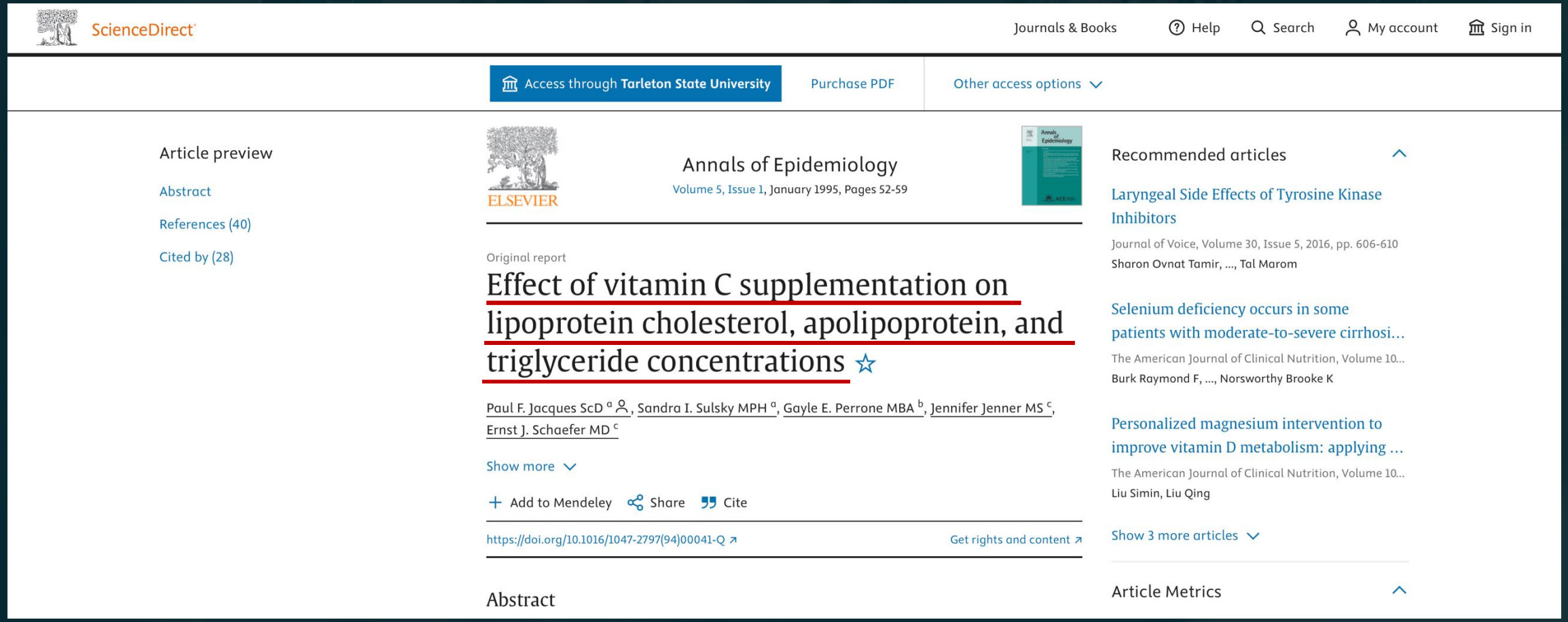

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Original report

Effect of vitamin C supplementation on lipoprotein cholesterol, apolipoprotein, and triglyceride concentrations ☆

Paul F. Jacques ScD [a], Sandra I. Sulsky MPH [a], Gayle E. Perrone MBA [b], Jennifer Jenner MS [c], Ernst J. Schaefer MD [c]

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Treatment Protocol

All study procedures were approved by the Human Investigations Review Committee of the New England Medical Center hospitals. Subjects were randomized with equal probability to receive either two 500-mg vitamin C tablets or two placebo tablets per day (one each morning and one each evening) for 8 months. Fasting blood samples were obtained at baseline, at 4 months, and at the end of treatment (8 months). We measured plasma AA levels and counted returned pills to assess compliance. For the present

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Although we saw no differences between treatment groups, there were differences between subjects in the baseline plasma AA tertiles. Subjects in the lower two tertiles were significantly less likely to be female and had significantly lower vitamin C intakes than did subjects in the highest tertile. Subjects in the lowest tertile also had significantly lower HDL cholesterol concentrations and higher LDL and total cholesterol concentrations, total/HDL cholesterol ratio, and fat intake.

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TABLE 3. Baseline characteristics of subjects who completed trial[a]

	All subjects		Baseline plasma ascorbic acid					
			< 55 µmol/L		55–79 µmol/L		⩾ 80 µmol/L	
	Vitamin C	Placebo	Vitamin C	Placebo	Vitamin C	Placebo	Vitamin C	Placebo
N[b]	74	64	22	21	23	20	29	21
Age (y)	41	41	43	42	42	38	39	43
Female (%)	38	39	32	24[d]	30	20[d]	48	71[e]
HDL cholesterol (mmol/L)[c]	1.19	1.18	1.12	1.17[d]	1.18	1.13[d,e]	1.26	1.27[e]
LDL cholesterol (mmol/L)[c]	3.08	3.26	3.28	3.65[d]	3.17	3.04[d,e]	2.87	3.02[e]
Total cholesterol (mmol/L)[c]	4.87	5.06	5.11	5.46[d]	4.87	4.75[d,e]	4.68	4.91[e]
Total/HDL cholesterol	4.1	4.3	4.6	4.7[d]	4.1	4.2[d,e]	3.7	3.9[e]
Apo A-I (µmol/L)[c]	32.1	32.2	29.6	31.0	34.7	31.8	32.7	33.7
Apo B (µmol/L)[c]	1.77	1.73	1.82	1.91	1.70	1.60	1.78	1.61
Triglycerides (mmol/L)[c]	1.17	1.22	1.41	1.28	1.08	1.19	1.09	1.18
Glucose (mmol/L)[c]	4.85	4.90	4.85	4.85	4.66	4.90	4.95	4.95
Body mass index (kg/m^2)	25.8	25.5	26.8	25.7	25.6	26.0	25.1	24.7
Fat intake (g)	65.8	67.9	69.6	74.8[d]	66.9	64.6[d,e]	62.1	63.5[e]
Vitamin C intake (mg)	134	118	97	90[d]	122	132[d]	179	144[e]
Nonwhite race (%)	12	8	9	10	22	11	7	5
Former smoker (%)	47	35	45	33	61	37	41	33
Alcohol intake (% > 10 g/d)	26	21	32	20	26	30	21	15
Postmenopause (% of women)	46	32	71	20	43	25	36	40

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Table 4 displays plasma AA concentrations by study period and treatment group. Mean plasma AA concentration in the placebo group was stable over the study period among subjects with higher baseline plasma AA concentrations, and rose slightly among subjects with lower baseline plasma AA concentrations. Plasma AA values in the vitamin C treatment group rose sharply and by month 8 were similar among subjects with higher and lower baseline plasma AA concentrations.

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TABLE 4. Mean plasma ascorbic acid levels (µmol/L) by treatment group[a]

	Baseline	Month 4	Month 8
All subjects			
Vitamin C	72	123	131
Placebo	67	68	71
Baseline plasma ascorbic acid tertiles			
< 55 µmol/L			
Vitamin C	39	113	125
Placebo	36	41	48
55–79 µmol/L			
Vitamin C	68	112	119
Placebo	68	76	77
⩾ 80 µmol/L			
Vitamin C	100	139	144
Placebo	97	89	88

[a] To convert to mg/dL, multiply by 0.0176.

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Table 5 presents the final lipid concentrations, adjusted for baseline lipid levels, as well as the change (± standard error) between the baseline and final measurements. There was no evidence for a vitamin C effect for any lipid measure among all subjects except for a marginally significant effect on apo A-I ($P = 0.08$). We observed that apo A-I concentration increased 1.9 μmol/L (5.3 mg/dL) with supplementation. There was a significant interaction between treatment and baseline plasma AA for HDL cholesterol ($P < 0.05$). When we stratified by baseline plasma AA, we observed a significant difference between treatment groups for HDL cholesterol levels ($P < 0.05$) and a marginally significant difference for total/HDL cholesterol ratio ($P = 0.08$) among subjects with a baseline plasma AA less than 55 μmol/L

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(1 mg/dL). The final HDL cholesterol levels were 0.10 mmol/L (3.8 mg/dL) higher in the vitamin C treatment group compared with the placebo group. Total/HDL cholesterol ratio was reduced by 8% among supplemented subjects with a baseline plasma AA level less than 55 µmol/L. The difference in apo A-I concentrations between treatment groups was also greatest (3.2 µmol/L or 9.0 mg/dL) among subjects with a baseline plasma AA level less than 55 µmol/L, but this difference was not statistically significant ($P = 0.11$). Stratification by baseline plasma AA did not reveal evidence of any vitamin C effects on other lipid parameters.

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Vitamin C Reduces Cholesterol-Induced Microcirculatory Changes in Rabbits

Anna Freyschuss, Rui-Juan Xiu, Jian Zhang, Xiaoyou Ying, Ulf Diczfalusy, Tomas Jogestrand, Peter Henriksson, and Ingemar Björkhem | AUTHOR INFO & AFFILIATIONS

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Abstract
Methods
Results
Discussion
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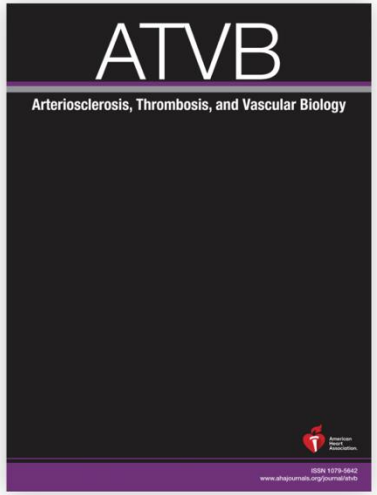
Abstract

Abstract The microcirculation was studied for 10 weeks in untreated rabbits (n=12) and in rabbits treated with vitamin C in their drinking water (0.5 g/d; n=6), a 1% cholesterol diet (n=12), or a combination of the two treatments (n=11). The studies were performed by direct intravital microscopic imaging of the conjunctiva of both eyes to evaluate blood flow velocity, microvessel diameter, and microhemorheologic conditions. As we reported previously, changes occurred in all of the aforementioned variables as a consequence of cholesterol feeding. After 3 and 6 weeks of feeding, there was a marked and significant (*P*<.0001) decrease in blood flow velocity in third-order arterioles, which was accompanied by stasis and erythrocyte aggregation in the smaller conjunctival vessels. When cholesterol treatment was combined with vitamin C, blood flow was almost identical to that of controls and significantly (*P*<.0001) higher than that of rabbits treated with cholesterol alone. All other changes were also



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Animals and Treatments

Forty-one young, male, New Zealand White rabbits with an average initial weight of 3.0 kg were housed individually under conditions of 12-hour light/dark periods. Twelve rabbits were fed standard chow and normal drinking water for 10 weeks. Another 6 rabbits received standard chow and drinking water supplemented with 500 mg of vitamin C daily. Twelve rabbits were fed standard chow supplemented with l% (wt/wt) cholesterol. A fourth group of 11 rabbits received standard chow supplemented with 1% (wt/wt) cholesterol and drinking water supplemented with 500 mg of vitamin C daily. The rabbit chow pellets were produced by and purchased from AB AnalyCen. The vitamin C supplementation consisted of tablets (C-vitamin sockefri, ACO) dissolved in the first bottle of water each day, after which time the rabbits were given unsupplemented drinking water ad libitum. Two separate sets of experiments were performed. One set of experiments included all 4 rabbit groups (6 rabbits from each group) and measurements at weeks 0, 3, 6, and 10. The second set of experiments included only 3 groups (control, cholesterol-fed, and cholesterol-fed and vitamin C–supplement rabbits) with n=6, 6, and 5 animals, respectively. For the second set of experiments, measurements were made at weeks 0 and 10 only. The group of rabbits treated with cholesterol and vitamin C originally contained 6 rabbits, from which 1 was excluded from study because this animal refused to eat the diet. At the end of the study, the rabbits were killed by an intravenous overdose of fluanizone and fentanyl. The project was approved by the Animal Ethics Committee (Stockholm, Sweden).

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Results

Lipid Levels

During the experiment the weights of the 4 sets of rabbits increased from an average of 3.0 to 3.7 kg. This increase in weight was slightly lower for both rabbit groups that were given dietary cholesterol (average final weight, 3.5 kg) than those fed the control diet with or without vitamin C (average final weight, 4.0 kg). Plasma lipid levels increased in the 2 sets of rabbits given cholesterol and cholesterol plus vitamin C ($P<.0001$), whereas no effect was seen on either cholesterol or triglycerides in the plasma of rabbits treated with vitamin C only. During the 10-week study, the rabbits that were fed cholesterol-supplemented pellets without vitamin C increased their cholesterol levels from 1.0±0.1 to 68±6 mmol/L ($P<.0001$) and triglyceride levels from 1.3±0.1 to 4.5±1.0 mmol/L (mean±SEM, $P=.01$). The rabbits that were fed cholesterol-supplemented pellets and vitamin C (in their drinking water) increased their cholesterol levels to as much as 63±6 mmol/L ($P<.0001$) and triglycerides to 3.9±1.2 mmol/L ($P>.05$). Thus, vitamin C treatment had no significant effect on either serum cholesterol or serum triglyceride levels ($P>.05$). Vitamin C also had no significant effect on serum cholesterol and triglyceride levels in rabbits that were fed the control diet ($P>.05$).

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Microcirculatory Changes

During the course of the study, several changes in the microcirculation were observed. It should be noted that 24 rabbits were investigated during weeks 3 and 6, whereas an additional 17 rabbits were investigated before treatment and after 10 weeks of treatment (see "Methods"). Among the microcirculatory variables, blood flow velocity can probably be regarded as the most reliable owing to the fact that accurate measurements can be made and relatively great effects obtained.[7]

As previously described,[7] blood flow velocity decreased markedly during treatment with cholesterol compared with the other regimens (P=.0009; Fig 1). This decrease was highly significant when compared with controls (P=.0002). The decrease was maintained through the sixth week of treatment but was attenuated after 10 weeks of treatment, possibly due to adaptive mechanisms. Blood flow, however, was still significantly lower in the cholesterol group than in all other groups. When cholesterol was combined with vitamin C treatment, blood flow was almost identical to that of controls and significantly (P<.0001) higher than that of rabbits treated with cholesterol alone. Vitamin C had no significant effect on blood flow in normolipidemic animals (results not shown).

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In accordance with previous work, arteriolar diameter also decreased significantly during cholesterol treatment compared with the other regimens ($P<.0001$; Fig 2). This decrease was also attenuated between weeks 6 and 10 and paralleled the changes in blood flow. After 10 weeks of treatment, there was still a significant difference in arteriolar diameter between the cholesterol-treated and the untreated rabbits ($P=.01$). Vitamin C treatment appeared to have a preventive effect on the cholesterol-induced reduction in arteriolar diameter after 3 and 6 weeks of treatment but not after 10 weeks of treatment (Fig 3). The difference in arteriolar diameter between rabbits treated with cholesterol and those treated with cholesterol plus vitamin C was highly significant ($P=.006$) after 6 weeks of treatment. Vitamin C had no significant effect on arteriolar diameter in normolipidemic rabbits.

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As expected,[7] cholesterol-fed animals showed increased stasis in their blood vessels compared with control animals (Fig 3). This stasis was maximal after 6 weeks of treatment and decreased slightly after 10 weeks of treatment. The changes in the cholesterol-treated rabbits were significantly different from those of control animals ($P<.003$). Vitamin C had a protective effect on cholesterol-induced changes, and the difference in stasis between rabbits treated with cholesterol and those treated with cholesterol and vitamin C was significantly different ($P<.007$ at 3 and 6 weeks and $P=.04$ at 10 weeks). Vitamin C had no effect on the occurrence of stasis in normolipidemic rabbits (Fig 3).

Cholesterol treatment also induced erythrocyte aggregation in microvessels whose diameters were <30 μm ($P=.0005$; Fig 4). In this case also, addition of vitamin C had a preventive effect ($P=.02$). Vitamin C had no effect on the occurrence of aggregation in normolipidemic animals (Fig 4).

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Discussion

The cholesterol-induced effects on the conjunctival microcirculation in the rabbit were the same as those demonstrated in our previous work, ie, reduced blood flow, decreased diameter of the arterioles, increased aggregation of erythrocytes, and increased stasis.[7] Blood flow velocity is the most reliable and sensitive quantitative microcirculatory parameter. It is noteworthy that the cholesterol-induced microcirculatory changes were not associated with marked changes in SBP. The preventive effect of vitamin C treatment on the cholesterol-induced changes in blood flow velocity was highly significant. As expected, relative changes in the diameter of the microvessels induced by cholesterol were smaller but still statistically significant. However, the results of these measurements also support the contention that vitamin C has a preventive effect on cholesterol-induced changes.

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It is well established that vitamin C is important for cholesterol metabolism in mammals (for a general review see Reference 17[17]). The role of vitamin C is most easily evaluated in species like guinea pigs, which are unable to synthesize vitamin C. Under normal conditions, species like rats and rabbits seem to synthesize vitamin C in sufficient amounts. Cholesterol feeding, however, may induce oxidative stress, and it has been shown that intake of exogenous cholesterol causes increased utilization of the vitamin.[18] These same studies have also shown that metabolic stress caused by dietary cholesterol can be counteracted by consuming larger amounts of vitamin C.[18] Very recently, it has been reported that utilization of vitamin C in guinea pigs is markedly enhanced by the combined effects of cholesterol and other constituents (eg, fat or sugar) of the Western diet.[19] The clear effect of vitamin C supplementation on the microcirculation of hyperlipidemic rabbits in the present study is consistent with an increased requirement for the vitamin under certain conditions. It has been reported in some[17] [20] but not all[21] studies that elevated serum cholesterol levels in rabbits after a load of dietary cholesterol is significantly reduced by vitamin C supplements. In the present study, administration of vitamin C had no significant effect on circulating cholesterol levels.

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The effect of vitamin C may be due to interference with either LDL oxidation or a process that is induced by oxidized LDL. A key factor controlling the rate of LDL oxidation seems to be its content of peroxy radical scavengers. It has been shown that 80% of the antioxidant capacity of the LDL particle can be accounted for by α-tocopherol.[22] There is, however, a synergistic interaction between vitamin C and α-tocopherol, and α-tocopherol can be regenerated from the corresponding radical by vitamin C.[23] [24] The resulting oxidation product of vitamin C, the semidehydroascorbate radical, can be converted back to the parent compound by the enzyme semidehydroascorbate reductase. In our study, vitamin C treatment had no significant effect on the levels of circulating vitamin E.

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Both water- and lipid-soluble peroxy radicals have been detected during LDL peroxidation, but the relative importance of these radicals for propagation of the chain reaction in the lipid phase of the particle is unclear. It has been suggested that vitamin C has an inhibitory effect on LDL oxidation by either scavenging peroxy radicals directly or acting synergistically with the α-tocopherol endogenous to the particle. In accordance with this scenario, vitamin C treatment has been found to increase LDL resistance to oxidation in vitro in rabbits.[25] Vitamin C deficiency is known to increase CCl_4-induced lipid peroxidation in vivo in guinea pigs,[26] and vitamin C supplementation decreases lipid peroxidation in vivo in rats that have an iron overload.[27] It has also been shown that vitamin C, in both its reduced and oxidized forms, may act as a preventive antioxidant against metal ion–mediated LDL oxidation.[28] Human subjects with low vitamin C levels have been reported to have higher amounts of lipid peroxides in plasma than do subjects with high vitamin levels.[24]

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Quercetin protects against atherosclerosis by regulating the expression of PCSK9, CD36, PPARγ, LXRα and ABCA1

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4.2. Quercetin's role in metabolic syndrome treatment

Quercetin modulates many processes related to the development of MetS, such as insulin sensitivity, lipid metabolism, and inflammation, as shown by network pharmacology research (30–32). The results align with previous studies indicating that quercetin's antioxidant and anti-inflammatory properties might be crucial in enhancing metabolic markers and reducing risk factors associated with MetS (33). Our research indicates that quercetin may enhance the benefits of a nutritious lifestyle, such as diet and physical activity programs, by interacting synergistically with other components.

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Abdominal obesity can be explained by the accumulation of excessive fatty acids and triglycerides in the liver and other organs (34), which results in organ dysfunction. Quercetin treatment can enhance abdominal obesity generated by monosodium glutamate by restoring imbalances in gut bacteria, which lead to damage in the hypothalamus and anomalies in retinol metabolism (35). Quercetin's potential to reduce obesity is associated with a decrease in fat accumulation and cell death by blocking the production of enzymes like MAPK and ACC. Increased expression of MAPK and its substrate, acetyl coenzyme A carboxylase, induces cell death and decreases the activation of extracellularly regulated and stress-activated protein kinases. Quercetin diminishes adipogenesis by stimulating the MAPK signaling pathway (36).

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Chronic high-fat, high-energy diets can damage the area of the brain that synthesizes acetylcholinesterase (37). Quercetin can reduce the effect of hyperlipidemia on memory impairment by affecting the cholinergic system in the nervous system, thereby increasing the activity of acetylcholinesterase (38). NF-kB activation contributes to an elevation in inflammatory factors, while quercetin suppresses NF-kB function and the production of inflammatory factors in pancreatic cells. ATP, a biological chemical pathway linked with damage, is generated in the vicinity of lysed cells and in high concentrations during infections. Quercetin can decrease hyperlipidemia and inflammatory processes by reducing ATP and ADP levels in the extracellular environment (39).

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Quercetin's advantageous effect on blood glucose levels may be due to many mechanisms, including boosting glucose uptake through GLUT4 and boosting glucokinase activity, promoting liver absorption of glucose, and suppressing liver glycogenolysis and gluconeogenesis (40), or alleviating oxidative stress and pancreatic β-cell damage, which can play a positive role in the control of diabetes mellitus.Akt, a serine protein kinase, can control adipogenesis and lipid metabolism via influencing LDL receptors. Quercetin has been apparent to stimulate the SIRT1 gene by increasing NAD+ levels. This regulates Akt function by enhancing deacetylation, shields β-cells from cytokine-induced negative consequences, and decreases diabetic nephropathy. Specifically, quercetin lowers glucose levels in diabetic rats' blood via boosting Akt and glycogen synthase kinase 3 phosphorylation (41). In

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5. Conclusion

Abdominal obesity, lipid metabolism abnormalities, and insulin resistance are among the diagnostic criteria for MetS, which is one of the eight components of life. According to the results of network pharmacology and molecular docking, quercetin may intervene in the MetS by participating in the AGE-RAGE signaling pathway in diabetic complications, lipids, and atherosclerosis as the main regulatory pathways; and according to the analysis of NHANES data, hypertension, diabetes mellitus, and body mass index may influence the correlation between LE8 and MetS, and it can be obtained that there is an inverse. As a result, we hypothesized that quercetin might regress MetS through food, blood glucose, and blood lipid control, therefore influencing the LE8 MetS score, which gives research ideas and references for quercetin's future development and clinical use.

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Modeling and grouping

A total of 54 12-week-old apoE$^{-/-}$ mice were divided into 3 groups using a random number table method (9) after being fed a regular diet for 1 week. The 3 groups included apoE$^{-/-}$ mice fed with a high-fat diet (model group, n=18), apoE$^{-/-}$ mice fed with a high-fat diet and treated with quercetin (quercetin group, n=18) and apoE$^{-/-}$ mice fed with a high-fat diet and treated with atorvastatin (atorvastatin group, n=18). The high-fat diet included 21% fat and 0.5% cholesterol, but otherwise was a normal diet. The C57BL/6 mice, at an identical age and strain, were fed with a normal diet and served as the control group (control group, n=18). The quercetin group and atorvastatin group received via oral gavage (once per day for 12 weeks), quercetin water solution (12.5 mg/kg) and atorvastatin water solution (2.06 mg/kg), respectively. The doses of quercetin and atorvastatin wer using a human equivalent dose formula based on 60 kg adults. The control grou group received via oral gavage, equal volumes of distilled water (once per day f

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General health conditions

The mice in the control group were in a good condition, with shiny coats and agile movements. The mice in the model group had become overweight, had coats with dry hair and slow movements. Compared with the mice in the model group, the mice in both the quercetin and atorvastatin groups were in a relatively better condition, with shinier coats and more agile movements. The mice in all the groups exhibited normal food consumption during the experimental period.

One-way ANOVA of repeated measurements revealed that there was no interaction between the groups and intervention time ($P>0.05$). The main effect caused by grouping and intervention time was statistically significant ($P<0.05$). The body weight of the model mice differed significantly from that of the control mice ($P<0.05$). Compared with the model group, significant differences were also observed in the quercetin and atorvastatin groups ($P<0.05$); however, the differences between the quercetin and the atorvastatin group were not significant ($P>0.05$). One-way ANOVA analysis revealed that the model group differed significantly at each time point (12-24 weeks) compared with the control group ($P<0.001$). The quercetin and the atorvastatin group exhibited significant differences compared to the model group ($P<0.05$) at week 17. The detailed data are presented in Fig. 1.

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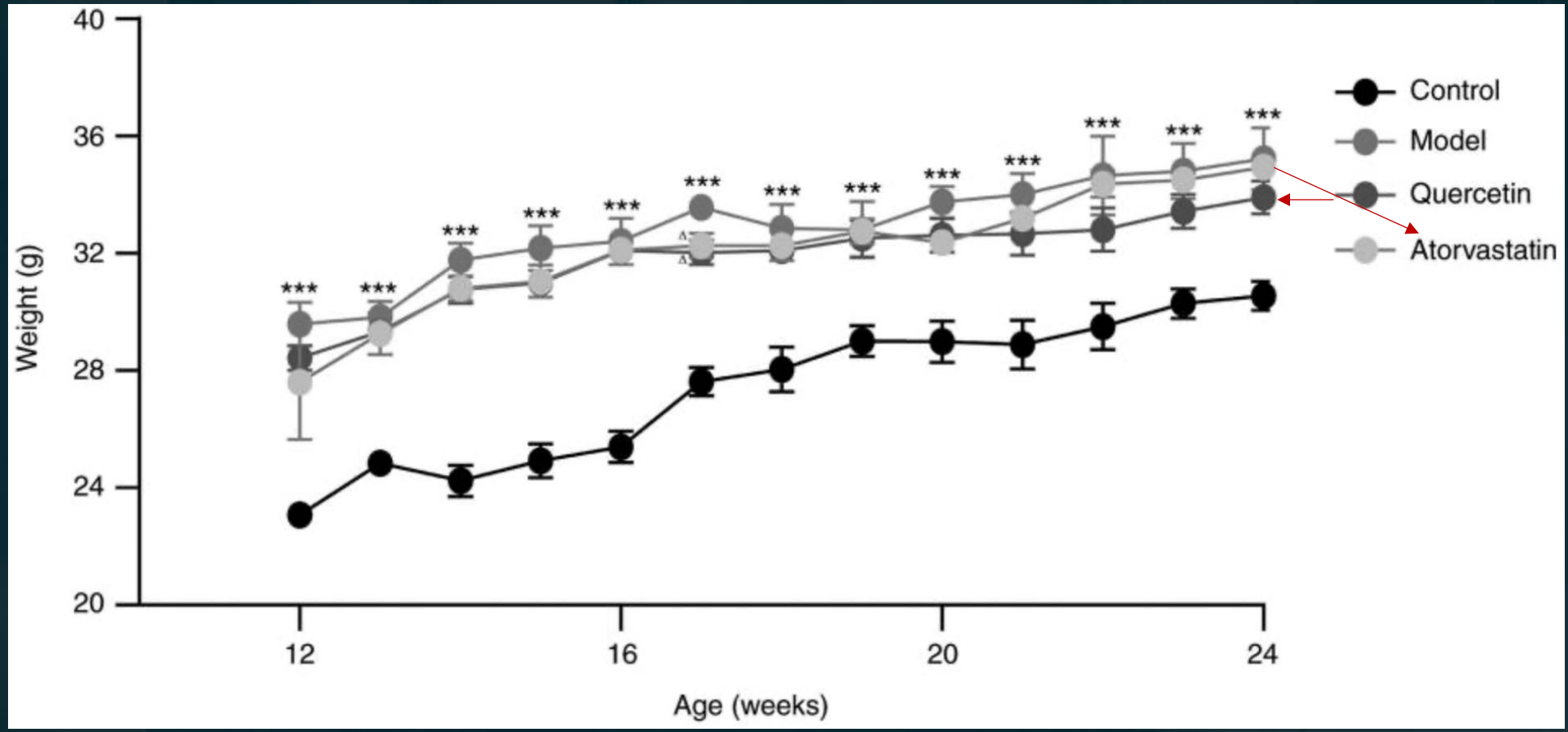

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Serum lipid profile

In the model group, the serum levels of TC, LDL-C and oxLDL increased significantly ($P<0.01$ and $P<0.001$), while those of HDL-C decreased significantly ($P<0.01$), as compared with the control group. The mice in the quercetin and atorvastatin groups exhibited significantly lower serum levels of TC, LDL-C and oxLDL than the mice in the model group ($P<0.05$, $P<0.01$, $P<0.001$). However, the differences between the quercetin and atorvastatin group were not significant ($P>0.05$). The detailed results are presented in Table I.

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Table I.

Comparison of serum lipid levels in each group.

Group	n	TC	TG	LDL-C	HDL-C	oxLDL
A	6	6.22±0.64	5.36±0.48	2.42±0.37	5.16±0.29	180.04±3.33
B	6	7.91±0.85[a]	5.77±0.32	3.15±0.46[a]	4.05±0.60[a]	242.46±3.93[b]
C	6	6.83±0.87[c]	6.13±0.45	2.48±0.41[d]	4.39±0.40	204.11±2.67[e]
D	6	6.46±0.83[d]	6.00±0.17	2.47±0.34[d]	4.41±0.78	199.24±4.29[e]

Data are presented as the means ± standard deviation. A, control group; B, model group; C, quercetin group; D, atorvastatin group.

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Pathological examination of mouse aortic tissue H&E staining

In the model group, a greater amount of atherosclerotic plaque was observed at the aortic root than the control group ($P<0.001$). Compared with the model group, the areas of atherosclerotic plaque in both the quercetin and atorvastatin groups were much smaller ($P<0.01$). However, the area of atherosclerotic plaque did not differ significantly between the quercetin and atorvastatin group ($P>0.05$). The detailed data are presented in Fig. 2.

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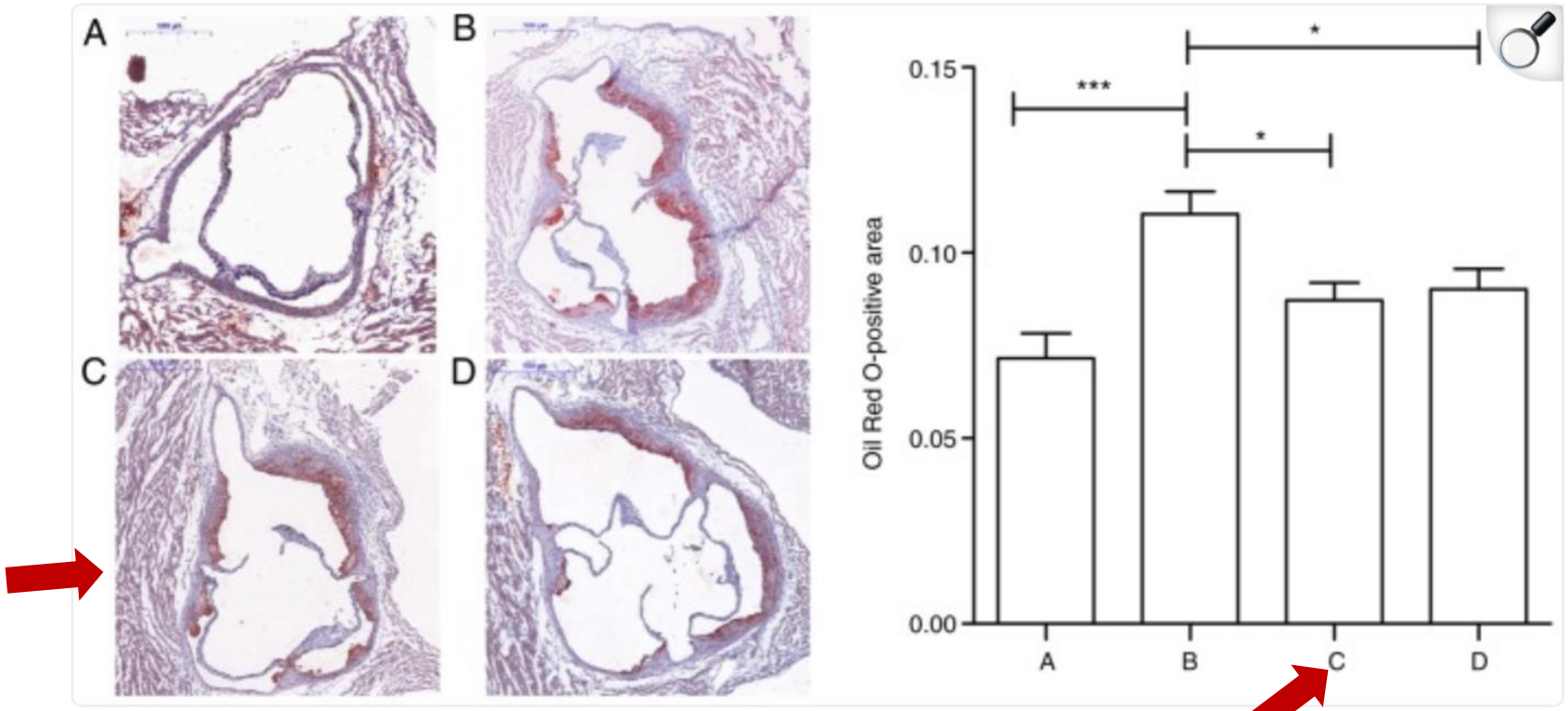

A, control group; B, model group; C, quercetin group; D, atorvastatin group.

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Quercetin is a flavonoid derived from plants including dodder, mulberry and others (27,28). Quercetin has a wide range of biological activities, such as cardiovascular protective effects, anti-atherosclerosis, anti-oxidation, anti-inflammation, increasing coronary blood flow, reducing capillary fragility and other effects (6). A previous study demonstrated that quercetin supplementation significantly reduces cardiac hypertrophy, a cause of cardiovascular morbidity and mortality (29). Recently, the mechanisms of the the anti-atherosclerotic effects of quercetin have been investigated extensively. For example, quercetin has been shown to effectively reduce the serum TC and LDL-C levels elevated by a high-fat diet in a rabbit model and has demonstrated antiatherogenic properties (30). Quercetin significantly reduces the endothelial expression of adhesion molecules induced by oxLDL, attenuated the TLR-NF-κB signaling pathway and thus decreases the inflammatory process in a rabbit model (31). In addition, quercetin has been shown to inhibit atherosclerotic plaque development in high fructose-fed C57BL/6 mice by inhibiting reactive oxygen species (ROS) production and activating the PI3K/ AKT signaling pathway (32). Quercetin significantly reduces lipid accumulation and the upregulation of CD36 mRNA and protein expression in oxLDL-activated RAW264.7 macrophage cells (33). Furthermore, quercetin protects macrophage from oxLDL-induced damage by regulating PCSK9 and ABCA1 expression, and thus prevents the progression of AS (7). In addition, quercetin regulates ABCA1 protein expression and increases the HDL-C level via the activation of the PPARγ-LXRα pathway, and consequently reduces the risk of atherogenesis (34,35).

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This study demonstrated that in apoE$^{-/-}$ mice fed with a high-fat diet, quercetin treatment significantly reduced the atherosclerotic plaque area, lipid accumulation and FC levels, and increased the collagen fibers in atherosclerotic plaques. In addition, quercetin treatment improved hepatocyte micro-structure, reduced the number of lipid droplets, as well as the Oil Red O-stained lipid content in liver tissue, and increased the liver collagen fiber content. Moreover, quercetin treatment resulted in increased serum levels of HDL-C and IL-10, together with decreased serum levels of TC, LDL-C, oxLDL, TNF-α and IL-6. Finally, quercetin treatment resulted in the downregulation of PCSK9 and CD36 protein expression and the upregulation of PPARγ, LXRα and ABCA1 protein expression levels in both the aortic and liver tissue. These results demonstrated that the atheroprotective effects of quercetin may be caused via regulating the expression of PCSK9, CD36, PPARγ, LXRα and ABCA1.

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The relationship between dietary magnesium intake, stroke and its major risk factors, blood pressure and cholesterol, in the EPIC-Norfolk cohort

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Briefly this sub-cohort (n=4,920) is comprised of a representative random sample of 4,000 men and women with complete data for food diaries from the EPIC-Norfolk cohort (n=25,369) and 1,102 stroke cases (n=182 part of 4,000 random sample previously mentioned) giving a total of 4,920. Participants were resident in the Norfolk area at recruitment between 1993 and 1997 and recruited through participating General Practices (n=35) (10). Participants were excluded from analyses if they had reported prevalent stroke at baseline or had missing values for any variables included in the multivariate model (n=477). Participants with missing values for smoking status, aspirin medication use for >3months, and magnesium from supplements (including medication) were recoded and classified as 'current smoking' (n=37) and 'no' aspirin (n=813) and 'no' supplements (n=2) to reduce the risk of bias due to under-reporting. Therefore 4,443 participants remained for analysis in this study.

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4.0. Discussion

The main findings of this case-cohort study of British adults suggest that, after adjustment for several important confounding factors including age, smoking status, history of CVD, medication use, total energy intake and other dietary variables, there was a strongly significant association ($P \leq 0.01$) between dietary magnesium intake and SBP and DBP in men, but not in women. There was also an association with TC in both men and women ($P=0.001$ and $P \leq 0.01$ respectively) which was attenuated but remained significant after adjustment for other dietary factors ($P=0.02$ and $P=0.04$ for men and women respectively). Furthermore in relation to stroke risk specifically, we identified a significant decrease in risk (HR 0.59 95% CI 0.38-0.93 $P=0.04$) in men with dietary magnesium intakes ≥ 354 mg/d (the highest 30%) compared to those with intakes ≤ 214mg (the lowest 10%).

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Compared with our findings, previous studies have shown a significant inverse association between dietary magnesium intake and SBP and DBP in men has also been reported (22) with differences of -6.4 mmHg and -3.1 mmHg for SBP and DBP respectively between those with the highest and lowest intakes in 615 older Japanese men (age 63-82 years) using 24hr recall. Using 7d food diary data, a more robust measure of dietary intakes, in a representative general population of middle and older age we identified slightly greater differences in BP between extreme quintiles of magnesium intakes with a difference of -7 mmHg ($P<0.001$) and -3.8 mmHg ($P<0.001$) for SBP and DBP respectively. We identified between quintiles differences were 250 mg/d for men, and 198mg/d for women, the equivalent to approximately 2 slices of wholemeal bread with peanut butter and 9 Brazil nuts, therefore achievable through dietary intakes (23). We also noted a tendency towards lower fruit, vegetable and bread and cereal intake in those with the lowest dietary magnesium intakes and may be relevant for identifying individuals whom may benefit from increased intake.

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In women a potential benefit from increased consumption of magnesium has previously been reported (24–26). Witteman et al (22) and Song et al (23) showed a reduction in relative risk RR 0.77 95%CI: 12%-33% and RR 0.93, 95% CI 0.86-1.02 respectively for developing hypertension in prospective studies using FFQs. Also a meta-analysis of RCTs using oral magnesium supplements also reported a dose dependent effect of supplementation on blood pressure (27). This is in contrast to the current study where we did not find any significant trends between magnesium intake and BP in women. This discrepancy may be due to differences in the models used. For example Witteman et al (1989) (24) did not adjust for lifestyle factors including physical activity levels and smoking status which are known to influence BP. We further explored why we might have identified differences between genders.

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It may be due to the fact that older age, higher BMI, and higher level of physical inactivity were more prevalent in women with low magnesium intakes which may in part explain why a significant effect was shown in men not women. As highlighted in the results section differences were identified between those with the lowest 10% of intakes and those with higher intakes, however, we took these factors into account during our analyses. It is also of note that differences identified were largely the same for both men and women, although there was a higher percentage of women, with the lowest 10% magnesium intakes, using antihypertensive medication which may influence the findings due to modifying effect of medication on future risk. In addition there was a narrower range of magnesium intakes for women (644mg/d) compared with men (744mg/d) which may attenuate the results. It may also be that the cohort was insufficiently powered to detect an affect in women.

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A number of intervention trials, using oral magnesium supplements, have reported significant reductions in BP ranging from 2.0 – 12.0 and 2.7 – 8.0 mmHg for SBP and DBP respectively (28–32). Although, the supplement doses were comparable with dietary intake, ranging between 200 mg/d to 600 mg/d, the formulations used were inconsistent.

Limited studies have previously investigated associations between dietary magnesium intake and TC or subfractions and two previous studies found no association with TC unlike our study which found that TC was ≈4% lower in Quintile-5 compared with Quntile-1 (33–36). However, a higher magnesium intake has been related to beneficial increases in high density lipoprotein (HDL) concentrations (33, 35).

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Magnesium has a number of metabolic roles in the body and may influence BP and blood lipids through different mechanisms (3, 4). Magnesium may serve as a natural calcium channel blocker, exhibit beneficial effects on platelet coagulation, have a potential role in vasodilation and has been associated with reduced coronary artery calcification (4, 5). Other proposed mechanisms include increased peroxidation of lipoproteins with subsequent acceleration of atherosclerotic plaque formation and low magnesium may facilitate an increase in inflammation which is associated with negative changes in lipid profile (3, 4). Higher magnesium intake has been associated with lower risk of Type II diabetes (6), metabolic syndrome (7) and cardiovascular disease (CVD) (8).

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Probiotic and selenium co-supplementation, and the effects on clinical, metabolic and genetic status in Alzheimer's disease: A randomized, double-blind, controlled trial

Omid Reza Tamtaji [a] · Reza Heidari-soureshjani [b] · Naghmeh Mirhosseini [c] · ... · Esmat Aghadavod [e] · Maryam Tajabadi-Ebrahimi [f] · Zatollah Asemi [e] ... Show more

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Summary

Background and aims

Combined probiotic and selenium supplementation may improve Alzheimer's disease (AD) by correcting metabolic abnormalities, and attenuating inflammation and oxidative stress. This study aimed to determine the effects of probiotic and selenium co-supplementation on cognitive function and metabolic status among patients with AD.

Methods

This randomized, double-blind, controlled clinical trial was conducted among 79 patients with AD. Patients were randomly assigned to receive either selenium (200 μg/day) plus probiotic containing *Lactobacillus acidophilus*, *Bifidobacterium bifidum*, and *Bifidobacterium longum* (2 × 10^9 CFU/day each) (n = 27), selenium (200 μg/day) (n = 26) or placebo (n = 26) for 12 weeks.

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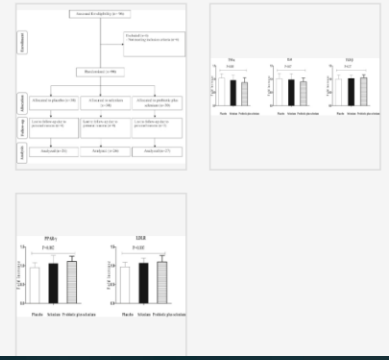

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3.2 Biochemical measurements

Selenium supplementation, compared with the placebo, significantly reduced serum hs-CRP ($P < 0.001$), insulin ($P = 0.001$), HOMA-IR ($P = 0.002$), LDL-cholesterol ($P = 0.04$) and total-/HDL-cholesterol ratio ($P = 0.004$), and significantly increased GSH ($P = 0.001$) and QUICKI ($P = 0.01$) (Table 3). Probiotic plus selenium intake resulted in a significant reduction in serum hs-CRP (−1.6 ± 1.4 vs. −0.8 ± 1.0 and +0.1 ± 0.5 mg/L, respectively, $P < 0.001$), and a significant increase in TAC (+89.4 ± 129.6 vs. +20.0 ± 62.5 and −0.7 ± 27.2 mmol/L, respectively, $P = 0.001$) and GSH levels (+122.8 ± 136.5 vs. +102.2 ± 135.2 and +1.5 ± 53.2 μmol/L, respectively, $P = 0.001$) compared with only selenium and placebo. In addition, subjects who received probiotic plus selenium supplements had significantly lower serum insulin levels (−2.1 ± 2.5 vs. −1.0 ± 1.3 and +0.7 ± 2.0 μIU/mL, respectively, $P < 0.001$), HOMA-IR (−0.5 ± 0.6 vs. −0.2 ± 0.3 and +0.1 ± 0.4, respectively, $P < 0.001$), and higher QUICKI (+0.01 ± 0.01 vs. +0.005 ± 0.007 and −0.002 ± 0.01, respectively, $P < 0.006$) compared with only selenium and placebo. Additionally, probiotic and selenium co-supplementation resulted in a significant reduction in serum triglycerides (−17.9 ± 26.1 vs. −3.5 ± 33.9 and +0.3 ± 9.3 mg/dL, respectively, $P = 0.02$), VLDL- (−3.6 ± 5.2 vs. −0.7 ± 6.8 and +0.05 ± 1.8 mg/dL, respectively, $P = 0.02$), LDL- (−8.8 ± 17.8 vs. −8.1 ± 19.2 and +2.7 ± 19.0 mg/dL, respectively, $P = 0.04$) and total-/HDL-cholesterol (−0.3 ± 0.7 vs. −0.4 ± 0.9 and +0.3 ± 0.6, respectively, $P = 0.005$) compared with only selenium and placebo. Probiotic and selenium co-supplementation significantly increased MMSE score ($P = 0.007$) and QUICKI ($P = 0.03$), and significantly reduced hs-CRP ($P = 0.01$), FPG ($P = 0.04$), insulin ($P = 0.04$) and HOMA-IR ($P = 0.02$) compared with only selenium.

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3.2 Biochemical measurements

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https://www.clinicalnutritionjournal.com/article/S0261-5614(18)32572-X/fulltext

4.4 Conclusions

Overall, the current study demonstrated that probiotic and selenium co-supplementation for 12 weeks to patients with AD had favorable effects on MMSE score, hs-CRP, TAC, GSH, markers of insulin metabolism, triglycerides, VLDL-, LDL-, total-/HDL-cholesterol, but it did not affect other biomarkers of inflammation and oxidative stress, FPG and other lipid profiles. In addition, co-supplementation improved gene expression of TNF-α, PPAR-γ and LDLR, but did not affect gene expression of IL-8 and TGF-β.

https://www.clinicalnutritionjournal.com/article/S0261-5614(18)32572-X/fulltext

Supplement Facts

Serving Size: 3 Capsules
Servings Per Container: 30

Amount Per Serving		% Daily Value
Vitamin C (as ascorbic acid)	1000 mg	1,666%
Citrus Bioflavonoids	1000 mg	*
Rutin	100 mg	*
Proprietary Blend	25 mg	*

Rose hips powder, acai berry extract, yellow pepper fruit extract, spleen powder.

*Daily Value not established

Other ingredients: rice flour, gelatin, vegetable lubricant.

Nature Wins Selenium (30 Count)

Nature Wins SKU: SELENIUM001--listing

$29.99

★★★★★ (8 reviews) + Write a Review

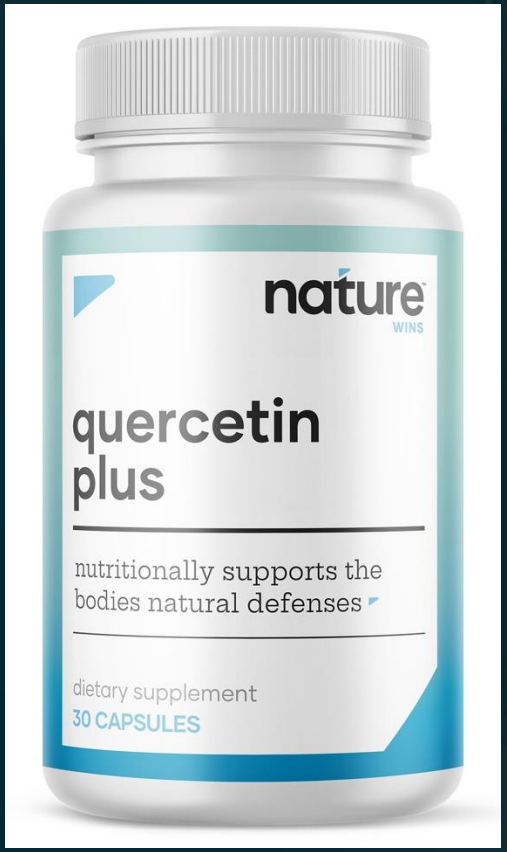
SIZE: *

1 Bottle
$29.99

3.00%
3 Bottles
$87.27

5.00%
6 Bottles
$170.94

https://thedrardisshow.com/



Supplement Facts

Serving Size: 1 Capsule Servings Per Container: 30

Amount Per Serving		% Daily Value
Quercetin	500mg	†

*Percent Daily Value (DV) are based on a 2000 calorie diet.
†Daily Value (DV) not established.

OTHER INGREDIENTS: Hydroxypropyl Methylcellulose (Vegetable Capsule), Vegetable Stearate.

Nature Wins Quercetin Plus (30 Count)

Nature Wins SKU: QUERCETIN001--listing

$29.99

★★★★★ (6 reviews) + Write a Review

SIZE: *

1 Bottle
$29.99

3.00%
3 Bottles
$87.27

5.00%
6 Bottles
$170.94

https://thedrardisshow.com/

Supplement Facts

Serving Size: 4 Capsules Servings Per Container: 30

Amount Per Serving		% Daily Value
Magnesium (as magnesium citrate, magnesium aspartate, and magnesium malate)	400mg	95%*

*Percent Daily Value (DV) are based on a 2000 calorie diet.
†Daily Value (DV) not established.

OTHER INGREDIENTS: Hydroxypropyl Methylcellulose (Capsule), Vegetable Stearate

Nature Wins Magnesium Complex (120 Count)

Nature Wins SKU: MAGCOMPLEX001--listing

$29.99

★★★★★ (49 reviews) + Write a Review

SIZE: *

1 Bottle	3.00% 3 Bottles
$29.99	$87.27
4.47% 6 Bottles	
$170.94	

https://thedrardisshow.com/

Supplement Facts

Serving Size: 1 Capsule Servings Per Container: 30

Amount Per Serving		% Daily Value
Zinc (as zinc picolinate)	30mg	200%*
Copper (as copper gluconate and sodium copper chlorophyllin)	2mg	100%*
Pea Powder	100mg	†
Millet Flour	50mg	†
Lentil Flour	50mg	†
Chlorophyll (as sodium copper chlorophyllin)	1mg	†

*Percent Daily Value (DV) are based on a 2000 calorie diet.
†Daily Value (DV) not established.

OTHER INGREDIENTS: Hydroxypropyl methycellulose (vegetable capsule), vegetable stearate.

Nature Wins Zinc Plus (30 Count)

Nature Wins SKU: ZINC001--listing

$19.99

★★★★★ (5 reviews) + Write a Review

SIZE: *

1 Bottle
$19.99

3.00%
3 Bottles
$58.17

5.00%
6 Bottles
$113.94

https://thedrardisshow.com/

A Comparative Study of Lipid-Lowering Effects of Guggul and Atorvastatin Monotherapy in Comparison to Their Combination in High Cholesterol Diet-Induced Hyperlipidemia in Rabbits.

Das S [1], Datta A [1], Bagchi C [1], Chakraborty S [1], Mitra A [2], Tripathi SK [1]

https://europepmc.org/article/MED/26735695

Abstract

Background

Hypolipidemic activity of gugulipid has been widely described in traditional literature.

Objective

This study was done to evaluate hypolipidemic activity of guggul and atorvaststin monotherapy in comparison to their combination in rabbits.

https://europepmc.org/article/MED/26735695

Materials and methods

Male New Zealand White rabbits (body weight 1.3-1.8 kg and age 8-10 weeks) were made hyperlipidemic by feeding cholesterol (0.5 g/kg) for three weeks and randomly divided into a control and three treatment groups receiving: atorvastatin (3.7 mg/kg), guggul (3.5 mg/kg) and their combination (same dose) for the next three weeks. Body weight measurements, estimation of serum lipid profile were done at the beginning, after three and six weeks, respectively. Histopathological examination of liver, heart and aorta was done after six weeks. Statistical analysis was done with SPSS version 16.0 using one-way and repeated measures analysis of variance (ANOVA) followed by post-hoc multiple comparison test with two tailed P value < 0.05 as significant.

https://europepmc.org/article/MED/26735695

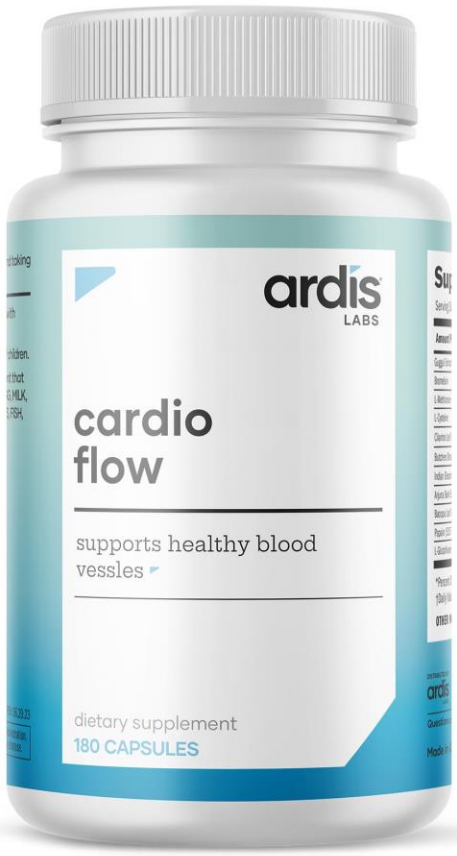
Results

All treated groups had significant reduction in cholesterol, triglyceride, low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) in comparison to pre-treatment values and control group, and had significant increase in high-density lipoprotein (HDL) in comparison to pre-treatment values.

Conclusion

Combination of atorvastatin and guggul was comparable to their monotherapies in improving lipid profile.

https://europepmc.org/article/MED/26735695



ArdisLabs Cardio-Flow (180 Count)

ArdisLabs SKU: CARDIO001–listing

$55.99

★★★★★ (4 reviews) + Write a Review

SIZE: *

1 Bottle	3.00% 3 Bottles
$55.99	$162.93
5.00% 6 Bottles	
$319.14	

ONE-TIME PURCHASE OR SUBSCRIPTION?: *

One Time Purchase	Ships every 30 days
Ships every 90 days	Ships every 180 days

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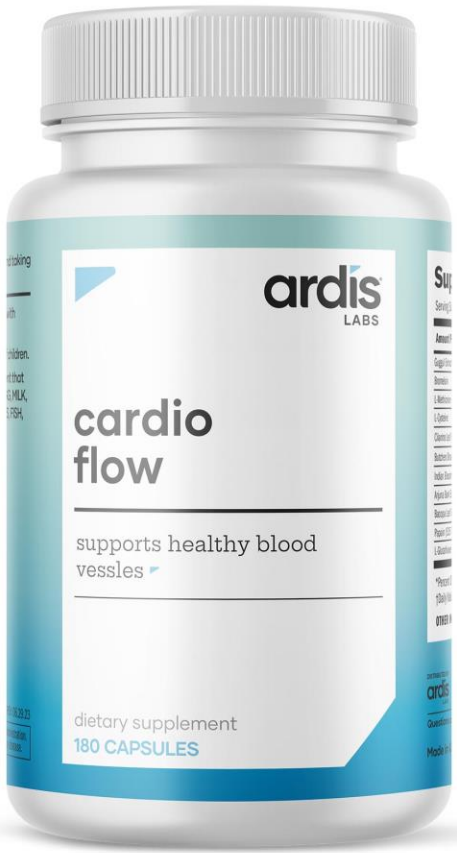

Supplement Facts

Serving Size: 2 Capsules Servings Per Container: 90

Amount Per Serving		% Daily Value
Guggul Extract (Std. to 2.5% Guggulsterones)	300mg	†
Bromelain	200mg	†
L-Methionine	200mg	†
L-Cysteine	200mg	†
Cilantro Leaf Powder	200mg	†
Butchers Broom Extract (Rhizome)	100mg	†
Indian Elecampane Extract 0.5% (Root)	76mg	†
Arjuna Bark Extract (Std. to 0.5% Arjunolic Acid)	50mg	†
Bacopa Leaf Extract (Std. to 20% Bacosides)	50mg	†
Papain (525 TU/mg)	20mg	†
L-Glutathione	14mg	†

*Percent Daily Value (DV) are based on a 2000 calorie diet.
†Daily Value (DV) not established.

OTHER INGREDIENTS: Vegetable Capsule (HPMC).

https://thedrardisshow.com/

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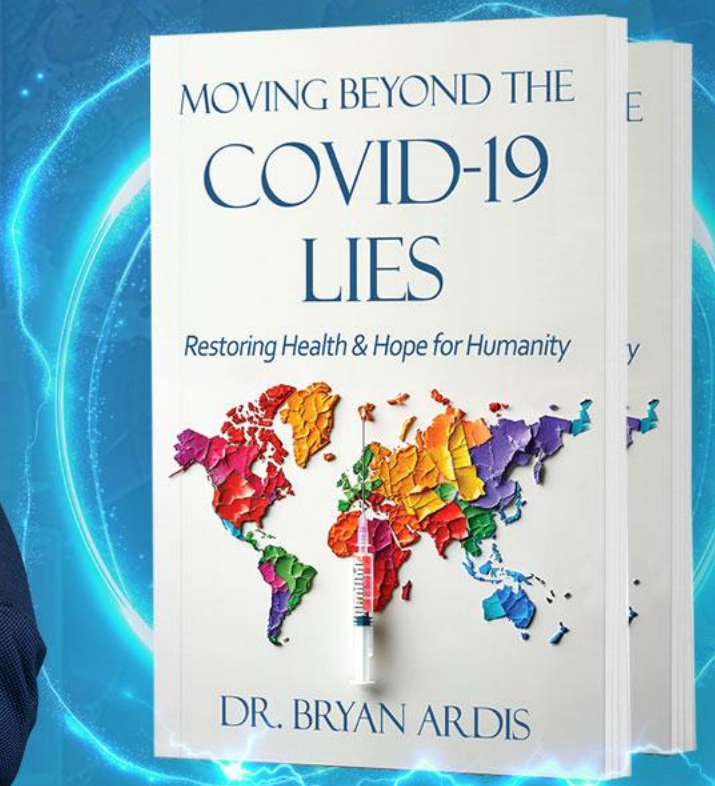
MOVING BEYOND THE COVID-19 LIES



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